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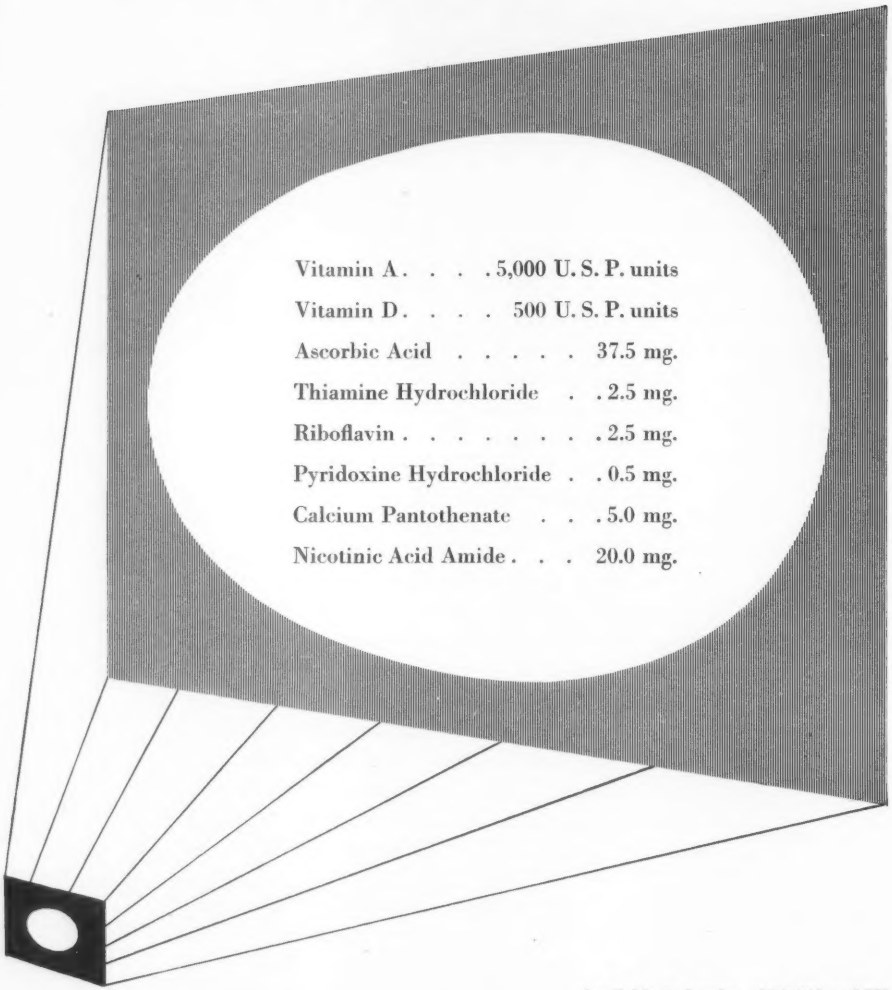
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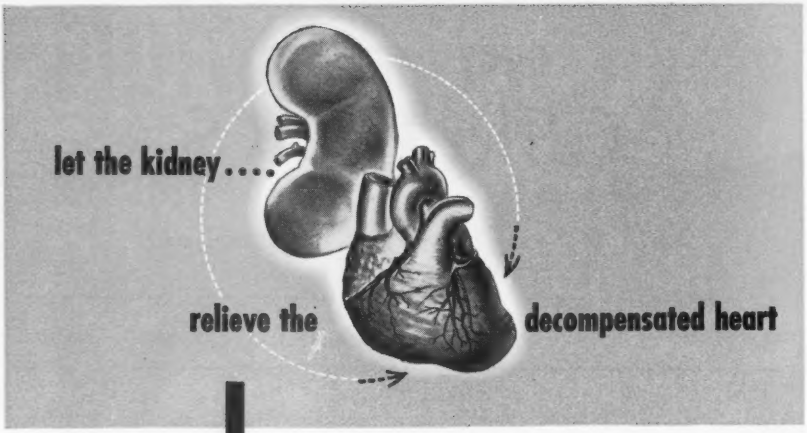

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ACUTE MYOCARDIAL INFARCTION

Mark D. Altschule, M.D.

Although myocardial infarction may occur in middle-aged or even young persons, it is evident that the disease is largely one associated with advanced age, for its occurrence parallels, in general, that of coronary sclerosis. The anatomical studies of Ehrich and others[1] and of Sappington and Cook[2] defined the changes in coronary arteries consequent to aging and showed how sclerosis occurs more rapidly with advancing years in the coronary than in the peripheral arteries. The past decade has seen a considerable increase in knowledge bearing on the pathological anatomy of coronary disease[3, 4, 5] and also on its electrocardiographic manifestations, but information relative to the physiological disturbances caused by infarction of a portion of the cardiac musculature is scanty. Most patients with the disease are much too ill to be subjected to extensive physiological study. Observations on animals are defective, in that operative exposure of the heart must be performed in order to induce infarction. Even though such studies may be carefully controlled, it is an error to assume that the effects of infarction of the heart can be estimated by subtracting the effects of anesthesia, operation, dehydration, and the like, from those of the procedure as a whole. Nevertheless, full appreciation of the clinical manifestations of any disorder rests largely on an understanding of the underlying physiological mechanisms, and it is, therefore, considered desirable to attempt an analysis of the various clinical syndromes exhibited by patients with acute myocardial infarction in the light of the known or probable disturbances of bodily functions which may occur.

Pain

Severe, protracted, constricting substernal pain is usually so striking as to overshadow other important manifestations. The pain of myocardial infarction is probably ischemic in origin. Lewis[6] showed that exercising a muscle under conditions of ischemia causes pain, an observation amply confirmed by other workers. The studies of Sutton and Lueth[7] on dogs, since corroborated by other investigators, showed that closure of a coronary artery gives rise to behavior consistent with pain. Whether the pain is a consequence of oxygen lack, damage to nerves running in the adventitia of coronary arteries, carbon dioxide excess, or the production of an excess of some metabolite is not known, although Lewis[6] favored the last. The

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production of cardiac pain by induction of anoxia in patients with coronary disease has been considered by some to be convincing proof of the anoxic origin of cardiac pain; however, breathing air containing low concentrations of oxygen not only induces anoxia but also increases cardiac work and gives rise to stimulation of the autonomic nervous system. It must be concluded that the origin of the pain of myocardial infarction is still obscure, although it appears that ischemia is important.

Afferent sensory fibers which respond to painful stimuli lie in the adventitia of the coronary arteries.[8] Fibers responsible for coronary pain apparently run through the middle and inferior cardiac nerves and the thoracic cardiac rami to the middle and inferior cervical and the first four thoracic sympathetic ganglia; thence they enter the spinal cord through the first four thoracic posterior roots.[9] These axones are small, poorly myelinated or unmyelinated, and their rate of conduction is slow;[10] they therefore resemble sympathetic motor neurones except that they run to the cord as a single fiber without any synapse in the ganglion between a pre- and postganglionic fiber. Other components of the pain of coronary disease, i.e. pain in the neck or jaw, are apparently carried in the vagus.[9] The entry of most of the pain-responding fibers into the cord in the first to fourth (or fifth) thoracic segments accounts for typical referred pain over the skin of the shoulder, arm, or inner fingers. The pain of pericarditis, often referred to the neck, may complicate the picture.

The pain of myocardial infarction usually responds well to the administration of morphia in doses of 15 to 30 mg.; demerol is also useful. At times morphia is without benefit, and in such cases oxygen may help. Oxygen, it should be noted, may be helpful in this connection even in the absence of pulmonary congestion or cyanosis. The biological activity of oxygen in the body does not depend directly on the amount combined with hemoglobin in the blood but depends on its pressure as a gas dissolved in the plasma. Thus inhalation of air enriched with oxygen increases the oxygen dissolved in the blood without necessarily increasing that combined with hemoglobin, especially if the latter is already normal, i.e., if there is no impairment of pulmonary function. In some instances stubborn pain of myocardial infarction, unresponsive to other therapy, may possibly be relieved by local anesthesia applied to sensitive points on the skin of the chest.[11]

Pre-existing Complaints and Precipitating Factors

The occurrence of myocardial infarction proves the existence of disease of the coronary tree except in rare instances. Nevertheless, many patients with myocardial infarction give no history of antecedent complaints. Only approximately half have had angina pectoris in the past, and less than half have a history of chronic congestive failure. In such diseases as diabetes and hypertension, myocardial infarction is especially frequent, possibly because of the high incidence of arteriosclerosis in these diseases. Of particular

interest among those with pre-existing anginal discomfort are the patients who present the syndrome of "premonitory pain." This complaint consists of exertional angina, angina decubitus, or atypical cardiac pain of recent onset, i.e., from a few days to as long as four weeks preceding the myocardial infarction. The origin of this pain is obscure; possibly it indicates the occurrence of partial occlusion of a coronary artery sufficient to cause ischemia of the involved myocardium; spasm of the coronary arteries also may play a part at this stage. The subsequent occurrence of the typical syndrome of myocardial infarction then indicates the death of the muscle. At any rate, it is clear that the recent onset of angina or of atypical cardiac pain is a strong indication for instituting bed rest for several weeks.

Precipitating factors include all those influences which either increase cardiac work or further decrease the supply of blood in the presence of reduction in available blood supply to the myocardium because of the narrowing of its vessels. Among those factors that increase cardiac work are effort, especially unaccustomed or unusually severe exertion, severe, protracted infection or emotional upset, and digestion and absorption of a large meal.[12] Factors precipitating myocardial infarction by decreasing blood supply to the heart include a fall in diastolic blood pressure due to spinal anesthesia, hemorrhage, or shock,[5] and spasm of the coronary arteries apparently consequent to distention of abdominal viscera,[13, 14] exposure to cold,[15] or other factors. Prevention of myocardial infarction in patients with coronary arterial disease therefore involves moderation in exertion, eating, exposure, and, where possible, emotion. Fall in diastolic arterial blood pressure to low levels should be prevented, and infections should be controlled as promptly as possible.

Shock

Shock is of common occurrence in patients with myocardial infarction, at times, it may precede the pain although usually it follows it. It most often occurs within the first few hours after the onset of pain, but, occasionally, especially in patients who develop pulmonary edema, its onset may not occur for a day or two. The nature of the shock of myocardial infarction is not clear. Its sudden onset and equally rapid offset suggest a neurogenic mechanism.[16] The blood volume, in contradistinction to traumatic shock, is not significantly decreased[17] unless severe pulmonary edema is also present. The venous pressure, however, is often low,[16, 17, 18, 19] which suggests a failure of peripheral mechanisms controlling the return of blood to the heart. Loss of tone of the small vessels has been reported.[20] The circulation time is normal or slow.[16, 17, 19, 21, 22] In a large majority of instances of early shock the shock is self-limited and terminates without deleterious consequences in a period of from one-quarter hour to two hours. Occasionally it lasts longer, and these cases have a poor prognosis. In a few cases infusions of plasma have helped; such infusions given slowly do not aggravate pulmonary edema if the latter is present.[23]

Cases of shock associated with pulmonary edema after myocardial infarction have a poor prognosis. Apparently, additional mechanisms act in such cases; these include anoxia and hemoconcentration,[17, 24, 25] the latter due to the loss of plasma from the blood as a consequence of its incarceration in the edematous lungs or extravasation into the air passages.

It should be borne in mind that syncopal attacks, which are caused by cardiac arrhythmia and not by a manifestation of shock, may also occur.

Hyperactivity of the Sympathetic Nervous System

Sympathetic hyperactivity develops early and is usually marked. It gives rise to cutaneous vasoconstriction as manifested by bluish pallor, palpably cold skin, and measurable reduction in peripheral blood flow.[26] Indeed, evidence of generalized vasoconstriction involving the rest of the arterial tree is also present. This generalized vasoconstriction tends to maintain the level of blood pressure, especially the diastolic, at a time when shock tends to make it fall. In some instances, seen shortly after the onset of the pain of the attack and before the development of shock, the arterial pressure may be found well above the normal level for those patients. The generalized vasoconstriction may also result in some decrease in renal blood flow. Other evidences of sympathetic hyperactivity are sweating and probably also the abdominal distention which are common after myocardial infarction; in the same category is the vesical paralysis which may become manifest in elderly patients.

Hyperactivity of the Parasympathetic Nervous System

The manifestations of parasympathetic hyperactivity which occur after myocardial infarction are many and varied. They include changes in function of the heart, lungs, and abdominal viscera. The cardiac manifestations include carotid sinus hypersensitivity[27] and also certain arrhythmias discussed below. As is discussed later in this article, cardiac asthma and probably pulmonary edema as well are also probably vagal in origin. The nausea, belching, and vomiting of myocardial infarction likewise may possibly be due to vagal hyperactivity. Atropine is usually not helpful in these conditions, for when given in doses large enough to be therapeutically effective, it usually produces side effects, such as excessive tachycardia, which are undesirable in patients in the acute stages of myocardial infarction.

Cardiac Arrhythmias

The occurrence of ventricular arrhythmias after damage to the ventricular myocardium is readily understandable. It is probable that in most instances the ventricular arrhythmias are the consequence of increase in irritability of the ventricular myocardium in the vicinity of the infarct. The most common disturbance in this category is the development of occasional extrasystoles. Many authorities strongly advise the use of quinidine in

patients who exhibit this disorder; it is, however, our equally strong feeling, based on the fact that this complication uniformly has been disregarded with no ill effects in patients under our supervision, that occasional extrasystoles require no treatment. On the other hand, the occurrence of frequent extrasystoles, especially if they arise from different foci, is an indication for the use of quinidine; this type of arrhythmia may be the forerunner of ventricular tachycardia or fibrillation. The occurrence of ventricular tachycardia, of course, calls for energetic treatment.

Although myocardial infarction in the vast majority of instances is limited to the ventricles, the common arrhythmias which follow it involve the auricle, or the sinuauricular or auriculoventricular nodes. These arrhythmias include sinus bradycardia, auricular standstill, sinus arrhythmia, shifting pacemaker, auricular premature beats, nodal beats, auricular fibrillation, flutter or tachycardia, and minor degrees of heart block; higher degrees of block, i.e. 2 to 1 or higher, are less common. Most of these are readily recognized as vagal in origin; evidence bearing on the vagal origin of the partial heart block and auricular flutter and fibrillation is presented in detail elsewhere.[28] These arrhythmias frequently may require no treatment since they usually disappear in a matter of minutes to hours in cases of auricular fibrillation and in hours to days in cases of heart block.[29] In the patient in whom auricular fibrillation or flutter is troublesome or persistent, either quinidine or digitalis is effective. It should be borne in mind that strong vagal influences giving rise to cardiac standstill of high degrees of AV block may cause syncope, Stokes-Adams attacks, or sudden death.

Febrile Reaction

The diagnosis of myocardial infarction and its differentiation from other types of cardiac pain depends upon the development of evidences of tissue necrosis; these consist of fever, leukocytosis, and acceleration of the sedimentation rate. It is important to remember, however, that fever is usually absent during the first day or two after the pain; indeed, subnormal temperatures may be found during the period of shock which often occurs initially. Even in the absence of shock, significant fever usually does not develop until twenty-four to seventy-two hours following the pain. The reason for this delay is not known. This characteristic of the temperature curve is useful in differentiating myocardial from pulmonary infarction, for in the latter the onset of the fever precedes, coincides with, or follows quickly the occurrence of pain in the chest. The rise in rectal temperatures after myocardial infarction is usually slight, but may reach 103°; it remains at that level with minor fluctuations for three or four days, and then falls gradually. In rare instances, the temperature may reach 104° or more in uncomplicated cases.

Leukocytosis of 12,000 to 16,000, but occasionally higher, frequently occurs. The sedimentation rate remains accelerated for a few weeks to sev-

eral months after the subsidence of fever and return of the leukocyte count to normal.[30] Persistent marked elevations of the sedimentation rate in the months following an episode of myocardial infarction suggests a poor prognosis.

The extent and duration of the rise in temperature in myocardial infarction cannot be used as an accurate guide to the size of the area involved. Large individual variations occur; in addition, some of the rise in temperature is probably a consequence of impaired heat dispersal via the skin secondary to vasconstriction. This phenomenon may lead to the occurrence of a cool skin when the patient is actually experiencing a fever.

Carbohydrate Metabolism

Hyperglycemia is common, and glycosuria is not rare in myocardial infarction. These changes are not to be construed simply as evidence of failure of glucose metabolism but rather as an indication of a change in balance between anabolism and catabolism. It has been shown that the blood lactate and pyruvate levels are increased after myocardial infarction[31, 32] in a manner similar to other forms of injury and fever, i.e., the changes are those of the "adaptation" or "alarm reaction" of Selye, and result from an increased rate of catabolism. In a general way, the severity of these changes, viz., elevation of blood sugar and of lactate and pyruvate levels, parallels the severity of the injury which causes them. It is, therefore, not surprising that as a rule glycosuria indicates a poor prognosis in nondiabetic patients with myocardial infarction.

The disturbance of carbohydrate metabolism which occurs has certain implications with respect to therapy also. The hyperglycemia of myocardial infarction does not call for the use of insulin, as it is self-limited, and indeed it does not respond well to insulin. In addition, if insulin is used, the variability in the state of carbohydrate metabolism may favor the occurrence of hypoglycemia, with markedly unfavorable consequences.[33]

It is to be expected also that diabetes, if already present, will be aggravated by myocardial infarction; glycosuria increases, and insulin becomes less effective. The treatment of diabetic patients after myocardial infarction requires meticulous supervision. In view of the undesirability of hypoglycemic reactions at this time, it is well to relax somewhat the control of glycosuria.

Protein Metabolism

As in the case of carbohydrate metabolism, protein metabolism also deviates in the direction of catabolism as a result of injury, including myocardial infarction.[32] Thus elevation of blood amino-nitrogen levels, negative nitrogen balance, and creatinuria appear.[32] The increase in protein catabolism contributes to nitrogen retention. When severe, this catabolic process may result in muscular wasting and persistent prostration. Efforts should be made to maintain nutrition after myocardial infarction.

Pulmonary Edema and Cardiac Asthma

Pulmonary edema or cardiac asthma are very common occurences after infarction of the myocardium, although the presence of the former may be overlooked at times. Most clinicians consider these respiratory syndromes to result from damage to the left ventricle, a concept at variance with the bulk of physiological evidence[34, 35] and also with the fact that infarction of the right ventricle also gives rise to pulmonary edema. Physiologists believe pulmonary edema to be due to reflexes activated by cardiac lesions and influencing the state of the pulmonary vessels.[34, 35] Certainly the reflex nature of the wheezing of cardiac asthma appears to be established.[36] Pulmonary edema, by causing anoxia and also by the withdrawal of fluid from the blood and its incarceration in the lungs, favors shock.

Whatever the mechanism of these syndromes, it is clear that in most instances they are self-limited or require only the use of morphia and oxygen for their control. Digitalis is of doubtful value, and atropine is not consistently helpful. Venesection, and even the use of tourniquets or positive pressure respiration, though temporarily of value in occasional cases, are potentially dangerous since they may precipitate shock. The coexistence of persistent shock and pulmonary edema indicates an unfavorable prognosis; in our experience infusion of plasma at slow rates, i.e., not over 5 cc. per minute, has not only not aggravated pulmonary edema but in a few instances has been followed by transient or permanent improvement.

Renal Involvement

Albuminuria, oliguria, and nitrogen retention are common after myocardial infarction, and their severity parallels, in a general way, the severity of the reaction to the injury; they are therefore of some prognostic value when persistent. The occurrence of shock gives rise to renal vasoconstriction and a marked decrease in renal blood flow. In addition, as has been pointed out previously, the accelerated rate of protein breakdown throws an increased burden on the kidney and may exaggerate the degree of nitrogen retention. This syndrome is usually self-limited and, at any rate, requires no treatment except the administration of adequate amounts of fluid by mouth.

Cardiac Decompensation

The manifestations of chronic congestive failure, including pulmonary congestion, venous engorgement, hepatic enlargement and peripheral edema may appear after myocardial infarction. Although they may be noted while the patient is still in bed, they not uncommonly become manifest only after the patient is up and about. In occasional instances congestive failure may last for several weeks and then disappear never to recur, apparently as a consequence of healing of the infarct. The early occurrence of congestive failure should, however, be regarded as a poor prognostic sign. Whatever

the course, the appearance of congestive failure should be treated with digitalis in full therapeutic doses, restriction of salt, and, when indicated, the use of diuretics. The concept that digitalis in therapeutic doses is dangerous in the presence of fresh myocardial lesions is not supported by careful observation.

Blood Clotting

Increased tendency toward intravascular clot formation is known to occur after myocardial infarction.[37, 38, 39] Factors responsible for this phenomenon include (a) slowing of blood flow consequent to shock, congestive failure, or oversedation; (b) increased viscosity of blood resulting from dehydration or loss of plasma into or through edematous lungs; and (c) changes in the clotting mechanism itself associated with injuries of all types.[40]

The tendency toward intravascular clotting is of obvious importance in the genesis of mural thrombi in the heart and thrombi in the veins of the legs. The administration of anti-coagulants appears to be of benefit in this respect, but until more is known regarding their action in patients with myocardial infarction, their routine use cannot be recommended.

Embolization

Embolization to the lungs from thrombi originating in the heart or in the veins of the legs is of common occurrence after myocardial infarction. Similarly, embolization to the brain, limbs, kidneys, spleen, or mesenteric vessels may also occur as the consequence of breaking-off of mural thrombi in the left ventricle. Usually only fresh thrombi, loosely adherent to the endothelium, give rise to emboli, but occasionally an old intraventricular mural thrombus, healed peripherally but necrotic centrally, may release fragments. Accordingly, embolization may be due to myocardial infarction and yet occur weeks or months later; such syndromes as those of cerebral vascular accident or infarction of an abdominal viscus at times may be the consequence of long-forgotten or even undiagnosed myocardial infarction.

Pericarditis

The occurrence of a transient pericardial friction rub is a helpful diagnostic sign in patients with obscure thoracic pain; unfortunately, this sign occurs in only a minority of instances. Aside from its diagnostic significance and the fact that its occurrence may modify the pain and distort the electrocardiographic pattern of myocardial infarction, this type of pericarditis is of no significance and has no sequelae.

Cardiac Rupture and Cardiac Aneurysm

Healing of a cardiac infarct is not complete until some weeks after the acute accident.[41] Soon after the occurrence of the infarct, when the in-

volved muscle is necrotic but not yet replaced by collagen, the ventricular wall is weakened; rupture of the heart may occur during the first two weeks of the illness. The symptoms of tamponade which follow are rapidly fatal. Marked restriction of movement should be the rule until healing of the infarct is adequate; i.e., at least three weeks after the infarction. If rupture does not occur, bulging of the weakened wall may develop with the formation of a thin-walled fibrous aneurysm of the left ventricle.

Some Deleterious Consequences of Treatment

The clinical manifestations of acute myocardial infarction are, of course, modified by therapy; at times, however, modification may not be in the direction of improvement. Thus morphia, even in doses as small as 15 mg. given subcutaneously, may give rise to nausea, vomiting, and itching intestinal motility may be depressed with resultant discomfort, gaseous distention, and difficulty in evacuation. Bed rest alone may also give rise to constipation, and, in elderly men, aggravate or cause the appearance of signs and symptoms of prostatism. The nausea, vomiting, collapse, tinnitus, fever, and electrocardiographic evidence of myocardial damage of quinidine intoxication may complicate the picture if this drug is used unwisely. Similarly, over-enthusiastic use of insulin in patients with glycosuria after myocardial infarction may, by causing hypoglycemia, be harmful.[83] The rapid injection of aminophylline may cause appreciable generalized vasodilatation[42] and so increase blood flow[43] as to throw an additional burden upon a heart which at the time is unable to stand it. The injection of pituitrin for the control of distention may precipitate severe cardiac pain, and adrenalin, administered for the relief of wheezes in cardiac asthma, may kill the patient. The use of high concentrations of carbon dioxide in an attempt to cure hiccoughs may also impose a large burden on the heart.

The sometimes fatal effect of the uses of enemata in patients with myocardial infarction should be stressed. As has been pointed out, many factors favor the development of constipation, which if not anticipated, or recognized early when responsive to mild laxation, may become so severe and distressing as to require enemata. The deleterious effects on the heart of distention of the gastro-intestinal tract are well known to physiologists;[13, 14, 44] clinical experience records the death of patients with myocardial infarction during administration of an enema. If an enema must be given, it should be warmed to body temperature and given slowly in small volume.

Sequelae of Myocardial Infarction

In most instances the patient may be regarded as recovered from the effects of acute myocardial infarction three months after its occurrence. It must be borne in mind, however, that sequelae occur in a small number of the recovered cases. Chronic congestive failure may appear or angina pec-

toris may become a problem; these should be treated in the usual way. It is also important to bear in mind the fact that in rare instances embolization to the brain, lungs, or abdominal viscera from a mural thrombus may occur for the first time months after myocardial infarction. Similarly, the development of the rare complication, aneurysm of the left ventricle, occurs after an interval of some months.

Some of the sequelae of myocardial infarction are poorly understood. For instance, persistent pain in the shoulder and arm, with vasomotor disturbances,[45] while not common, does occur and is troublesome; there is no acceptable explanation for its occurrence. Profound weakness and easy fatigability together with mild emotional depression occur often; aside from the fact that this syndrome resembles the state seen after serious operations or severe infections, little is known about it. It constitutes a therapeutic problem. Benzedrine, in doses of 5 to 10 mg. by mouth, may be helpful; hypertension or recent myocardial infarction dos not constitute contraindications to its use in these doses.[46]

The common emotional sequelae of myocardial infarction include not only the mild, self-limited depression of a prolonged convalescence but also the development of severe anxiety neurosis. This is understandable, for cardiac pain itself is usually associated with a feeling of impending disaster which may impress the patient more than does the pain. In addition, some of the implications of myocardial infarction, i.e., sudden death, or the loss of ability to support himself financially, may cause a persistent feeling of anxiety in the susceptible patient, with palpitation, weakness, tachycardia, sweating, dyspnea, and cardiac pain. The cardiac pain may either be a sharp, sticking pain under the left nipple, as is characteristic of anxiety, or a squeezing substernal pain, i.e., true angina, induced by emotional stress in a patient with coronary disease. At times, the visceral manifestations of anxiety may be present without a verbal expression or even a conscious recognition of that emotion, a situation which may lead to confusion in diagnosis when interrogation of the patient is hurried or superficial. The importance of the physician's role in the precipitation or the prevention of this syndrome after myocardial infarction cannot be stressed too greatly. Too rigid restriction of activity, leading to invalidism; failure to provide for substitution of interests for those given up, leading to boredom and introspection; and frightening bluntness where reassurance is needed—all these may combine to favor the development of neurosis. Also to be borne in mind are the effects of the patient's illness on the family, i.e., financial depletion, curtailment of social life, and similar changes, which may prove disturbing to the family and so be reflected on the patient. In occasional instances, the patient's emotional reaction to his previous myocardial infarction and the invalidism which follows it may take the form of a severe depression with an attitude of hopelessness regarding rehabilitation, deep feelings of unworthiness consequent to inability to perform his familial offices, and a suicidal tendency.

Conclusions

Myocardial infarction gives rise to extensive physiological changes and consequently a wide variety of clinical syndromes. Appreciation of the existence and nature of these phenomena is helpful in the rational management of patients with infarction of the myocardium.

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MEDICAL RESEARCH GRANTS RECOMMENDED FOR APPROVAL OF SURGEON GENERAL

MEDICAL Research grants amounting to more than two million dollars have been recommended for the approval of Dr. Thomas Parran, Surgeon General, United States Public Health Service, by the National Advisory Health Council. Grants are contingent upon appropriations by the Congress for the fiscal year 1948 which begins July 1, 1947.

Grants recommended for approval for research studies in gerontology are as follows: Dr. Gregory Pincus, Worcester Foundation, Massachusetts, $10,844, Dr. Maurice B. Visscher, University of Minnesota, $19,710. Dr. H. S. Liddell, New York College of Agriculture, $34,247, Dr. Henry S. Simms, Columbia University, New York, $10,850. *Federal Security Agency.*

DIET FOR THE AGED

Jacob Meyer, M.D.*

WHAT should old people eat? How much, what kind, how many calories, how much protein, carbohydrate and fat? These questions are asked by physicians, dietitians and the general public. The present interest in the problems of the aged has focused the attention of those interested to the diet and nutrition of the aged. Much of what is practiced in the field of dietetics for the aged is based on the tradition or customs of the people. This is perhaps true to a degree for the diet of any age. There is much fear and prejudice about foods, not only by the aged person but by the relatives, the housekeeper, or the Home for the Aged. Some foods are regarded as bad for the aged. Certain foods are considered as poorly digestible or others cause constipation, diarrhea, etc.

While it is well for the physician, the dietitian and the public to respect the traditional food customs and habits of a people at any age, it is perhaps equally important to inquire into the scientific data which may help us formulate the answers to many questions asked concerning the diet of the aged.

CALORIC REQUIREMENTS

What are the caloric requirements of the normal aged? The known facts are rather limited. Benedict[1] found that the minimal heat production in women over 60 years of age is about 1000 calories every 24 hours, irrespective of the weight or height. In a series of measurements upon women between the ages of 60 and 83, the total heat production varied from 799 to 1540 calories.

Thewlis[2] states that the amount of food required in senescence varies with the individual. This author quotes Stiebling and Phipard "that the average man whose height is 68 inches, weight, 154 pounds and age, from 60 to 75 years requires 2700 calories a day when he is moderately active and 2160 calories when he is doing sedentary work. Past 75, the requirements are 10 per cent less.

"For the average woman whose height is 64 inches, weight, 132 pounds and age, from 60 to 75 years, 2250 calories are required when she is working moderately and 1800 calories when she is doing sedentary work." (These studies are from diets of families of employed wage earners and clerical workers in the cities of the United States). McCay[3] directs attention to the extensive studies of Fenger on a woman who lived for a period of 15 years on a very frugal diet. The daily caloric allowance varied from 1125

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to 1600. This diet served to maintain body weight which fluctuated between 42 to 45 Kg. On the other hand McCay refers to a table of diets in institutions for the aged by Forster, 1877. (See Table) The present day diets of institutions for the aged are rather high in calories. The proportion of the carbohydrate is also rather high.

TABLE
DIETS FOR THE AGED IN EUROPEAN INSTITUTIONS IN 1877 (FORSTER)

	Diets Consumed by Aged People			
	Protein	Fat	Carbohydrate	Calories
Munich Women	79	49	266	1871
Munich Men and Women	92	45	332	2157
Brandenberg	98	28	501	2962
Schwerin (Schwerin)	92	40	502	2772
London	70	32	341	1983

It is thus seen that there is a variation in the opinions of many as to the energy requirements of the aged; also, that the caloric intake approximates that of the normal adult, at rest and at work.

Clinicians of experience are familiar with the fact that the aged may maintain weight and good health, irrespective of caloric intake, whether low or high. The nutrition of the aged is apparently dependent on factors other than the number of calories eaten. It is probable that tissue dehydrations, delayed tissue repair may be significant factors in weight loss. Dubois[4] has summarized the data on basal metabolism of old people up to 1936. There is a definite drop of the average rate of basal metabolism with age, the decline being more rapid in the male than in the female sex, but "there is a great deal of variation in old people, probably depending on the degree of senility."

It would seem then, on the basis of the literature, that it is difficult to state the exact caloric requirements of the aged, and that since the data available approximates that of the normal adult diet, the diet for the aged should be that of the normal adult. For purposes of budgeting the food requirements of an institution for the aged, or the indigent aged, it would seem wise to use the normal adult caloric requirement as a standard, plus allowances for the cost of extra amount of vitamins. The problem of undernutrition in the aged is not solved by the diet per se. The factors of tissue dehydration and metabolism mentioned above are important. Equally important perhaps are the emotional and psychic factors. Severe or radical alteration in the diet of the aged may produce emotional disturbances which may lead to serious nutritional changes. On the other hand, social maladjustments among the aged, such as insecurity and dependency, may result in such depression that they will not eat, thus resulting in undernutrition.

CARBOHYDRATES, PROTEINS AND FATS

There is much confusion as to the use of carbohydrate, protein and fat in the diet of the aged. Some of this is fear, based on the idea that carbohydrate in the aged increases the danger of diabetes, that protein increases

hypertension, favors albuminuria and increases renal damage, and that fats promote arteriosclerosis. In the dietary studies by Fenger,[3] the distribution of foods (Case 1) was 1.95 gms. protein, 0.53 gms. fats, 3.7 gms. carbohydrate per Kg., (Diet No. 4); protein 2.3 gm., F. 1.94 gm., C. 3.6 gm. per Kg.

Chemical balance studies were made by Koch[3] on 5 men, ranging in age from 54 to 79 years. These men were living in the almshouse in Helsingfors, Finland. They were fed simple diets of meat, potatoes and vegetables. They consumed an average of 556 gms. of dry stuff, 245.3 gms. of water, 106 grams of protein, 55 gms. of fat and 34 gms. of ash. Of the food consumed they utilized an average of 86 per cent of the protein, 92 per cent of the fat and 94 per cent of the carbohydrates and 92 per cent of the calories.

It will be noted that carbohydrates comprise a greater proportion of the food of the aged. The absence of teeth or a poorly fitting set of teeth is the reason that they often select a soft, mushy diet, which consists mostly of carbohydrate foods. It is also said that a great many favor sweets. My associates and I have studied carbohydrate digestion[5] of the aged. Although we have found that salivary ptyalin is diminished, nevertheless, 80 per cent of the carbohydrate ingested is converted into dextrin in the stomach. Pancreatic amylase, according to our studies, is not diminished and we believe that the pancreatic amylase completes the digestion of carbohydrates. There are very few if any symptoms in old people indicating faulty carbohydrate digestion. Where the pancreatic secretion is altered we may expect some difficulty. We have found that maltase or taka diastase may be of value when there are clinical symptoms of incomplete carbohydrate digestion. The aged may also require an extra amount of vitamin B, because of the high carbohydrate content of the diet. Diabetes among them is most generally arterosclerotic in origin. Restriction of carbohydrate because of the fear of diabetes, seems unwarranted. It is wiser to permit them to have carbohydrates and spill some sugar, than subject them to the distress of weighed and measured diets plus insulin.

Protein digestion depends on the gastric and pancreatic secretions. In our studies[6] on the gastric secretions of the aged, we observed a decrease in the volume of secretion, acidity and pepsin. The only exception reported in this respect is a greater psychic secretion of gastric juice in the aged. This secretion is of low acidity as contrasted with the high acid secretion of young individuals. Thus the greater psychic secretion in the aged will not help gastric digestion, but on the contrary dilutes the subsequent gastric and hormonal phase of gastric secretion. We therefore must reckon with the possibility of a diminished protein digestion in the stomach. The stimulated pancreatic secretion in old subjects shows that trypsin concentration is unchanged compared to a young control group. The question arises whether a normal trypsin secretion can make up for a decreased pepsin

secretion and hydrochloric acid in the digestion of proteins. We know that tryptic digestion is enhanced by predigestion with acid pepsin or hydrochloric acid. The aforementioned chemical balance studies by Koch[3] would seem to indicate utilization of 82 per cent of the proteins. Further complete balance studies are needed to confirm these observations. Our clinical experience also leads us to believe that the average person digests proteins well. We are of the opinion that the protein per kilogram in the aged should approximate the amount given to a normal adult. We do not regard it necessary to seriously alter the amount of protein which an old person has been taking and generally advise no drastic change in the eating habits of older individuals. It should be obvious of course that where the proteins have been deficient, and where there is clinical evidence of hypoproteinemia, proteins can be supplied to the aged in the normal form or as amino acids. The fear that protein intake may increase renal damage or cause increased tension should not be taken too seriously.

It is general experience that the aged eat but little fats. The amount of fat in the diets by Forster are rather low. In our studies on pancreatic secretion we found that the concentration of lipase was considerably diminished. This has led us to suggest that moderate portions of fat are advisable in the aged.

The evidence from the literature and our own studies indicates that carbohydrates, proteins and fats are digested by the gastric, intestinal, and pancreatic secretion, and that they are apparently almost completely utilized by the chemical processes in the aged.

VITAMINS

The entire problem of vitamins for the aged requires a more complete study. Sebrell[7] recommends the daily adult allowances for the aged, vitamin A 5000 IU, vitamin B 1.8 mg., vitamin C 75 mg., riboflavin 2.7 mg. and niacin 18 mg. We have suggested that because of the greater intake of carbohydrate, the aged require more vitamin B than normal. Rafsky and Newman[8] suggest that the absorption of thiamine in the aged is inadequate and recommend supplementing the diet with additional vitamin B. These investigators have also studied vitamin C retention and excretion in the aged, and determined the saturation level. Large doses were required in all their patients (with the exception of two) in order to obtain a saturation level.

Deficiency syndromes in the aged may occur because of poor eating habits, poor absorption from the intestinal tract, or a combination of both. The symptoms of senility, such as dryness of the skin, loss of hair, edema, glossitis, diarrhea, constipation, tremor, mental impairment, are symptoms which occur in vitamin deficiency. It is important for the clinician to differentiate both conditions, but it is well to remember that both vitamin deficiency and age may be the cause of the symptomatology. The relationship of vitamin deficiency to the aging process, or to premature senility is not

clear as yet. It is significant that some of the mental symptoms of the aged have been reported as favorably relieved by ample doses of nicotinic acid.[10]

MINERALS

Decalcification, which occurs in the aged directs attention to the need for further studies on calcium in this group of people. Calcium and phosphorous relations are certainly important. The question of calcium absorption from the intestinal tract is significant and in this respect any disturbances in fat absorption would have a significant bearing on this problem. It is not uncommon to find that milk and cheese and other high-containing calcium foods are recommended for the aged.

ALCOHOL, TOBACCO, COFFEE

There is too much prohibition and proscription of these substances and I can see no serious objection to their moderate use. It must be recognized that the stimulating effects of alcohol on gastric juice is of digestive value, and the general vascular stimulation or relaxation caused by alcohol is equally valuable. In the private home, as well as in an old people's home, a good glass of wine or an ounce of whiskey or brandy is recommended for those whose custom it has been to partake of these beverages. Beer or ale is likewise recommended. Tobacco should be permitted to both sexes. Tea and coffee in moderation are likewise advised. Why deprive the aged of what little pleasure they may enjoy?

GENERAL RECOMMENDATIONS

In general, it is recommended that the diet of the aged should not be seriously changed from the normal habits and customs of the individual. It is recognized that the aged may live and maintain good nutrition on a relatively low caloric diet. In general it is our belief that the majority will eat enough calories to supply their energy needs. There are some who eat too much, and others who do not eat enough. This is true for any age, and in such instances, clinical evidence of overnutrition or undernutrition will become manifest and can be adjusted by the physician. If a psychic, emotional, or mental factor is discernible as the cause of abnormal eating habits, an attempt should be made to correct the underlying cause. It is probably wise to supplement the diet with extra amounts of vitamins, particularly B and C. Institutions for the aged, in planning the caloric needs of their residents, may be guided by the caloric requirements of the normal adult and the same standard should be applied for budgetary allowance for the aged indigent who are not in institutions. My associate, Dr. H. Sorter, who has worked very closely with old people, informs me that among the very poor, on admission to a home for the aged, there is a great desire for eggs every morning. This, she says, is not due to the actual desire for the egg, but to the belief among these people that the "rich" have eggs for breakfast

every morning and now they would like to have the same feeling of luxury.

Numerous basic diets for the aged have been suggested by many authors. The basic diet recommended by Sebrell for the elderly in normal nutrition is sensible and adequate and is repeated below:

"One pint of milk or its equivalent—much of this may be utilized in creamed soups, puddings, milk toast and as cheese.

One serving of orange, grapefruit, tomatoes or other juices.

One serving of green or yellow vegetables.

One serving of potatoes or other vegetable or fruit.

One serving of whole grain cereal, oatmeal or wheat.

One egg daily, or 3 or 4 a week.

One serving of meat, poultry, seafood, or other high protein food.

All bread and flour should be enriched or whole grain.

Butter, oleomargarine with vitamin A, peanut butter or other vitamin rich fats are indicated."

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GERIATRIC STUDIES IN BUENOS AIRES

A course in geriatrics recently given at the Hospital Britanico, Buenos Aires, under the auspices of the Junta Argentina para el Estudio del Envejecer, included the following lectures: Dr. B. A. Houssay, Physiology of Old Age; Dr. E. B. del Castillo, Endocrinology in Old Age; Dr. A. C. Tanquini, Geriatrics of Cardiovascular System; Drs. A. Casanegra and C. A. Cardini, Geriatrics of Respiratory and Gastrointestinal Systems, respectively; Dr. F. E. Kraupf, Neuropsychiatry in Old Age; Dr. D. del Valle, Geriatrics and Surgery; and Dr. A. Trabucco, Urogenitary Surgery in the Old. *J.A.M.A.*, March 22, 1947.

THE CLINICAL DIFFERENTIATION OF SENILE AND ARTERIOSCLEROTIC PSYCHOSES

David Rothschild, M.D.

WITHIN recent years there has been increasing recognition of the fact that great numbers of old persons are being admitted to hospitals for mental disease. During the year ending June 30, 1945, 41.9 per cent of all patients entering the Worcester State Hospital for the first time were in the age range extending upwards from sixty years.[1] Two well known disorders, namely, senile psychoses and psychoses with cerebral arteriosclerosis, account for most of these cases. According to state-wide figures for 1938,[2] the latter condition alone is responsible for more first admissions to hospitals for mental disease in Massachusetts than any other psychosis.

The magnitude of the problem emphasizes the need for accuracy of diagnosis in mental disorders affecting elderly persons. Unfortunately, mistakes occur frequently, not only in differentiating between senile and arteriosclerotic psychoses but in distinguishing them from a variety of other mental conditions. This is probably due to the fact that our clinical conceptions of these two disorders are apt to be hazy and inexact. They undoubtedly resemble each other in many ways, but they also exhibit numerous points of deviation. In the present paper, which is based on data published in earlier communications,[3, 4] their differences as well as resemblances will be stressed in an attempt to show that they can be delimited sharply enough to permit greater accuracy in diagnosis.

Since senile and arteriosclerotic psychoses present features in common and may even occur together, one may tend to think that there is little need for their careful differentiation. Such a tendency should be combatted, not only for scientific reasons but also for considerations of a practical nature. The two conditions possess different pathologic processes, and it is by no means certain that the same etiologic factors are operative in them. These points have been discussed elsewhere[3]. Their clinical courses are usually

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dissimilar, with consequent differences in the immediate prognosis. If they are not separated from each other our clinical description of each disease will be lacking in clarity, thereby increasing the danger of confusion with mental illnesses of an entirely diverse nature.

Definition and Classification.—The term senile psychosis or senile dementia should be used for those chronic mental disorders occurring in elderly persons and presenting a variety of clinical pictures which show in common progressive signs of organic mental impairment in association with characteristic senile changes of the brain. Senile psychoses are classified into the following types: simple deterioration, depressed and agitated type, delirious and confused type, presbyophrenic type, and paranoid type. The designations of the first three types are self-explanatory. Simple deterioration is the commonest form, accounting for about fifty per cent of the whole senile group. In the presbyophrenic type, the patient exhibits, in addition to the usual signs of intellectual enfeeblement, a marked tendency to relate false stories (fabrications) to fill in memory gaps, a jovial mood, loquacity and aimless restlessness. Typical forms of this condition are uncommon. In the paranoid form of senile psychosis, well developed and more or less persistent delusions are in the foreground; they may be of a persecutory, sexual or grandiose nature, alone or in combination, and are often accompanied by hallucinations of similar content. The intellectual faculties are apt to be much better preserved than in the other types. However, it should be emphasized that a clear-cut delimitation of these types is frequently impossible, particularly the first three mentioned, since transitions and admixtures of all sorts are common.

Arteriosclerotic psychoses include those acute and chronic mental disorders of middle-aged and elderly persons associated with focal damage to the brain caused by cerebral arteriosclerosis. Here, too, a variety of mental disturbances along with organic mental impairment is observed, but the symptoms do not fall into patterns warranting special clinical subdivisions.

Sex Distribution and Frequency.—Senile psychoses are nearly three times commoner in women than in men, and arteriosclerotic psychoses are approximately three times commoner in men than in women[4]. As to overall frequency, arteriosclerotic disease accounted for 15.1 per cent and senile dementia 14.0 per cent of all first admissions to State hospitals for mental diseases in Massachusetts[5] during the year ending June 30, 1945.

Age of Onset and Duration of Illness.—In my material the age of onset of senile dementia ranged from the sixties to the nineties, the average being 75 years, and the illness lasted from 7 months to 11 years, with an average duration of 4.7 years. In the arteriosclerotic group the age of onset ranged from the middle forties to the eighties, the average being 66 years, and the duration of the illness varied from one day to 16 years, with an average of 3.4 years. Thus, the mental illness tends to begin later and to last longer in senile dementia than in arteriosclerotic psychoses, and the latter

show greater variations in the duration of the disorder. In about 25 per cent of the arteriosclerotic group the psychosis terminated fatally within less than six months, whereas none of the senile patients presented such a short illness.

Manner of Onset.—In both groups the mental illness is usually preceded by a gradual diminution of physical and mental capacity consistent with normal old age. Senile patients tend to show this a little more frequently and in a more outspoken form than arteriosclerotic patients. In about 25 per cent of the senile cases, mental or physical stresses seem to act as precipitating factors. These factors include deaths in the family, financial difficulties, somatic disease and the like. Such factors are less common in arteriosclerotic cases.

Senile psychoses begin gradually except in occasional cases complicated by arteriosclerotic disease. Psychoses with cerebral arteriosclerosis may also begin gradually, but an abrupt onset is noted in a little more than half the cases. This is commonly, but by no means invariably, associated with gross neurologic evidence of a cerebral insult, such as hemiplegia or aphasia.

Early Symptoms.—Regardless of the manner of onset, practically all arteriosclerotic patients show one or more of the following features: headache, dizziness, syncopal attacks, convulsive seizures, symptoms of cardiac decompensation, and apoplectiform attacks. These disturbances are often noted for months or even years before any mental changes develop, but they may occur later. They are not ordinarily encountered in senile cases.

In arteriosclerotic cases with a sudden onset the disorder manifests itself as a confused state or delirium which does not differ essentially from the confused and delirious pictures observed in a great variety of severe somatic diseases. When the illness begins gradually the mental symptoms are similar in many ways to those of senile dementia. In both conditions there is slowly increasing intellectual failure, as indicated by loss of efficiency, impairment of memory, errors of judgment and the like. An exaggeration of previous personality traits is occasionally noted as an early feature. Sleeplessness and restlessness are common. Irritable, quarrelsome and jealous behavior, sometimes leading to threatening and violent acts are often in the foreground. In some cases loss of interest and apathy are prominent. Deterioration of personal habits and lowering of moral standards may be observed.

On the other hand, the two disorders exhibit certain differences. Arteriosclerotic patients often complain bitterly of weakness, fatigue and a great variety of unpleasant somatic feelings; depressive feelings and fear of failing health are common, and pronounced suicidal tendencies are by no means infrequent. Outspoken disturbances of this type are seldom encountered in senile cases. Emotional instability with easily provoked outbreaks of weeping or laughter is not uncommon in arteriosclerotic patients, but does not occur in senile patients. While delusions and hallucinations

may be noted in both conditions, persistent and well elaborated delusional ideas are generally observed only in senile psychoses, constituting the paranoid form of that disorder. Fabrications and jovial, restless behavior, especially if pronounced, are more likely to occur in senile dementia.

Course of the Illness.—In the well developed senile psychosis the organic mental impairment is obvious. There is diffuse impairment of memory, though recent impressions are more severely affected than remote ones. Orientation is defective for time and place. Impairment of comprehension is noted, and speech becomes rambling and incoherent. These disturbances grow steadily more marked, though the rate of progression varies greatly from case to case. In my material the patients had been ill for periods ranging from five months to ten years before it became necessary to hospitalize them. As a rule, the paranoid form of senile psychosis differs from the other types in that the intellectual disturbances are at first minimal, perhaps even difficult to detect, and may remain relatively mild for years. However, unless death occurs in the earlier stages from extraneous causes, all senile patients eventually present the familiar picture of a helpless, vegetative state associated with profound impairment of all mental activity. Such patients generally succumb to pneumonia, infections from bed sores, or other terminal infections.

In arteriosclerotic disease, psychoses which progress slowly are also observed, but an uneven and stormy course is more frequent. These patients may enter the hospital within hours, days, or weeks after the outbreak of mental symptoms. Improvement is common, but acute episodes can occur at any point in the course of the illness. They range from transitory clouded states to severe delirious or confused reactions which persist for weeks or even months. These episodes are sometimes associated with neurologic signs of a cerebral insult. They may terminate fatally or they may subside, leaving the patient with a varying amount of intellectual defect, but occasionally leading to a good remission which may last for months or years. The impairment of memory and of intellectual functions in general is usually spottier and less severe than that observed in senile dementia. In most cases, however, repeated attacks occur, and there is an increasing residue of mental deterioration during the quiescent periods. Even if acute episodes are lacking, the mental symptoms tend to increase and occasionally a picture of advanced mental decay not unlike that of senile psychosis is noted in the late stages of the illness. Death usually occurs from a cerebral accident, arteriosclerotic heart disease or a terminal infection.

Neurologic Changes.—In both psychoses tremors, changeable muscular rigidity and an uncertain gait are frequently observed. While more striking changes are not encountered in uncomplicated senile cases, the majority of the arteriosclerotic patients show obvious neurologic signs, though not necessarily early in the disease. The commonest type of alteration is hemiplegia or hemiparesis which may ante-date the psychosis, mark the onset, or occur

late in the course of the illness. Aphasic disturbances, and less frequently, pseudobulbar palsy or a picture suggestive of paralysis agitans may be noted.

Other Somatic Changes.—Changes customarily associated with old age, such as wrinkling of the skin, wasting, arcus senilis and the like, are usually present in senile dementia. Similar alterations are common in arteriosclerotic psychoses but are seldom pronounced except in cases with senile admixtures. In both psychoses the peripheral arteries almost always exhibit sclerosis and the blood pressure is often elevated. High figures occur only a little more frequently among arteriosclerotic patients, though they are more likely to show increased diastolic pressures than the senile patients. Thus, in 31 anatomically verified cases of senile psychosis, the systolic pressure was below 150 in 12 cases, between 150 and 200 in 13, and above 200 in 6 cases, with an average of 165 systolic and 83 diastolic for the whole group. Despite such figures, outspoken cardiac hypertrophy is uncommon in senile patients. In 29 anatomically verified cases of arteriosclerotic psychosis, the systolic pressure was below 150 in 7 cases, between 150 and 200 in 17, and above 200 in 5 cases, with an average of 172 systolic and 95 diastolic. In contrast to the senile group, patients with arteriosclerotic psychosis often show marked cardiac hypertrophy, coronary sclerosis, and attacks of cardiac decompensation.

Laboratory Observations.—Urinary changes are common in both psychoses; apart from extraneous disease, they are usually of a minor nature. They tend to be more severe in the arteriosclerotic patients who not infrequently show an increase of the nonprotein nitrogen content of the blood. Such findings do not ordinarily occur in senile patients except as a terminal phenomenon. Changes in the urine and blood indicative of diabetes are occasionally observed in arteriosclerotic but not in senile patients.

Differential Diagnosis

Differentiation Between Senile and Arteriosclerotic Psychoses.—Although most of the differences and resemblances between the two disorders have already been mentioned, it seems desirable to review them in order to stress the features which are of diagnostic aid as well as those which may be misleading.

In the average case of senile psychosis, the insidious onset in later life and the slow but inevitable increase of mental and physical decay extending over a period of years provide a picture that can scarcely be mistaken for any other condition.

Similarly, in a considerable number of cases arteriosclerotic psychoses present an easily recognizable picture with headache, dizziness and apoplectiform phenomena in the foreground. However, it should be emphasized that neurologic changes are often lacking in the earlier stages of the illness and they may never occur. In such cases there may be syncopal attacks, bodily alterations of a senile nature suggest a senile psychosis rather than

convulsive seizures or cardiac attacks. In my experience, convulsive seizures are by no means uncommon in cerebral arteriosclerosis (so-called arteriosclerotic epilepsy), but they are not observed in senile dementia. The importance of cardiac involvement in arteriosclerotic psychoses should be stressed, almost half of my cases showing attacks of cardiac dysfunction at one time or another. Since similar attacks are not ordinarily encountered in senile dementia, their diagnostic value is obvious.

In comparison to senile dementia, the course of arteriosclerotic mental disease shows greater diversity from case to case and more variable symptoms in the same case at different times. The disorder may consist solely of a confused state associated with a cerebral vascular accident, leading to a rapidly fatal outcome or constituting merely an evanescent mental disturbance; *or it may present a number of acute episodes which punctuate quiescent periods or remissions. At the other extreme, one occasionally sees chronic arteriosclerotic psychoses with a gradual onset and much less obvious fluctuations in the severity of the symptoms. In these cases, a clue to the diagnosis may be provided by early subjective complaints, anxiety concerning impending mental failure, and even outspoken suicidal tendencies, as features of this type are seldom prominent in senile dementia. Also, the arteriosclerotic patient usually shows a more uneven and less severe defect of memory than the senile patient, whose intellectual faculties are in general more seriously disturbed.

If neurologic accompaniments are lacking, the sudden attacks of confusion typical of many arteriosclerotic cases cannot readily be differentiated from the chronic confused states observed in senile dementia, unless a history of their development is available or their tendency to clear up reveals their arteriosclerotic basis. In senile psychoses, certain phenomena may come and go, such as restlessness, agitation, emotional reactions, hallucinations, and delusions, but the general intellectual impairment continues on its downward course.

In the paranoid form of senile psychosis, the intellectual faculties may be relatively well preserved and this may suggest an arteriosclerotic psychosis. In my experience, however, persistent delusions which are elaborated into a more or less well connected group of ideas rarely occur in arteriosclerotic disease, though ideas of mistreatment, jealousy and the like are not uncommon as isolated or temporary symptoms.

A reliable differentiation between the two disorders cannot be made solely on the basis of the blood pressure or the condition of the peripheral arteries. The fact is, peripheral sclerosis is just as common in one as in the other, and both disorders present a wide range of blood pressures. However, an increased diastolic pressure is more likely to occur in arteriosclerotic psychoses. Severe renal or uremic complications point to the

*Such conditions, which are undoubtedly common, are technically psychoses, even though they may be of little practical importance from a purely psychiatric viewpoint.

latter unless they are terminal phenomena. General wasting and other arteriosclerotic disease, especially if they are pronounced. Of course, arteriosclerotic mental disease often affects persons who are below the age range of senile psychoses, but otherwise the age of onset in the two disorders shows too much overlapping to be of definite diagnostic value. Similarly, the greater frequency of senile psychoses among women and of arteriosclerotic psychoses among men is of little help in the individual case. In my experience, carcinomatous growths are not uncommon in senile patients but are rare in arteriosclerotics, while diabetes is occasionally noted in the latter but not in the former.

Mixtures of Arteriosclerotic and Senile Psychoses.—From a purely anatomic viewpoint, mixtures of senile and arteriosclerotic changes are common. As a rule, however, one or the other process predominates and the clinical picture conforms largely to the predominant condition, so that obvious clinical admixtures are not observed with great frequency. A senile psychosis complicated by cerebral vascular disease may be revealed by slight fluctuations in the severity of the intellectual disturbances and less frequently by gross neurologic changes, such as hemiplegia, occurring in a setting of diffuse mental decay and a generally steady downhill course. In arteriosclerotic psychoses with a senile component, the illness tends to affect older patients and the distinctive arteriosclerotic features are accompanied by unusually severe mental deterioration or pronounced physical alterations of a senile nature.

Diagnostic Problems Involving other Mental Disorders.—Middle-aged and old persons are subject to a number of mental disorders which may be mistaken for senile or arteriosclerotic disease. The common sources of error will be discussed in the following paragraphs.

Psychoses associated with a great variety of somatic disorders and toxic conditions may be difficult to distinguish from arteriosclerotic disease since they usually present a mental picture similar to that observed in many cases of cerebral arteriosclerosis, namely, a sudden attack of confusion or delirium. If neurologic evidence of cerebral vascular disease is lacking, these two groups may be differentiated by discovery of the underlying toxic or somatic factors in the one group, and by the characteristic prodromal and early arteriosclerotic mental symptoms in the other. In psychoses due to the cardiac or renal disease, the difficulties are apt to be especially pronounced because disturbances of the heart and kidneys may be important accompaniments of arteriosclerotic psychoses. In such cases it is largely an arbitrary matter to estimate the relative importance of each factor in the total picture.

Similar diagnostic problems may be presented by diabetes. This disease is sometimes held responsible for certain mental disturbances, such as depressive reactions and confused states. On the other hand, diabetes

is often associated with arterial disease and the latter may lead to an arteriosclerotic psychosis. A true diabetic psychosis will respond to diabetic therapy and not follow the course of an arteriosclerotic mental disorder.

Surprisingly enough, psychoses due to toxic or somatic disease are not infrequently mistaken for senile dementia, even though the onset and course are obviously dissimilar in the two groups. Old persons are probably more vulnerable to toxic influence than younger ones, and the underlying somatic disorder may not be readily discernible. In my experience, however, the chief source of error is an uncritical attitude whereby one tends to forget that old persons may suffer from a great variety of psychoses. As a result of such an attitude, one may neglect to look for other possibilities, so that almost any form of mental disturbance may be labelled as a senile psychosis if it happens to occur at an advanced age, or as an arteriosclerotic disorder if it falls within a somewhat earlier age range.

Paranoid conditions of non-organic origin may resemble paranoid types of senile dementia. In the former the onset is usually earlier, and in the latter there should be evidence of organic intellectual defects, even if only slight.

Late involutional depressions may be confused with the depressed and agitated form of senile dementia. In the latter the depression is more superficial and often consists largely of a state of apprehension without clear motivation. Confusion may also arise with arteriosclerotic mental disease in which severe depressive reactions are occasionally observed; here the occurrence of somatic disturbances typical of cerebral vascular disease may be of diagnostic aid. Of course, the conclusive point of differentiation from both disorders is that involutional depressions are not associated with defects of memory, orientation and the like.

Certain forms of neurosyphilis may present diagnostic difficulties. General paresis occasionally occurs in elderly persons and may then be mistaken for senile dementia. Grandiose and euphoric traits or pupillary abnormalities may provide clues pointing to general paresis, but in some instances serologic study of the blood and spinal fluid may be necessary to establish the correct diagnosis. Vascular forms of neurosyphilis may resemble arteriosclerotic psychoses so closely that their differentiation will depend on a similar serologic study.

The so-called presenile psychoses, namely, Alzheimer's disease and Pick's lobar atrophy, are relatively rare conditions which resemble senile dementia in many ways, but they usually begin at an earlier age, in the forties or fifties. They may bear a superficial similarity to arteriosclerotic mental disease. They differ from the latter in that they display a gradual onset with diffuse intellectual impairment in the foreground, their course is steadily progressive without any sudden attacks of confusion and gross neurologic changes do not occur, though minor ones may be observed.

Cerebral embolism may produce a psychosis which, if it occurs in the

same age group, cannot be distinguished on the basis of the mental symptoms from an arteriosclerotic condition. This is not surprising since it causes essentially the same type of cerebral damage, namely, focal lesions. Thus, its diagnosis will depend on discovery of the underlying etiology, usually endocarditic vegetations from which the emboli originate.

Mistakes are occasionally made in differentiating chronic subdural hematoma and brain tumor from arteriosclerotic mental disease. In these cases a careful review of the manner of development of the symptoms or the presence of signs of increased intracranial pressure, such as vomiting and choked discs, should reveal the correct diagnosis or at least the need for additional neurologic study, including possibly electroencephalography and ventriculography.

In conclusion, it may be stated that despite a variety of pitfalls in the clinical diagnosis of senile and arteriosclerotic disorders, the two conditions show sufficient differences to render their separation feasible. Furthermore, they can be differentiated from other disorders if one remains alert to the fact that mental illnesses which are neither arteriosclerotic nor senile are common in elderly persons. It is not claimed that all mistakes can be avoided. Nevertheless, it is believed that a correct diagnosis can be established in the great majority of cases by a critical review of the whole picture without recourse to anything beyond ordinary clinical procedures.

Summary

Senile psychoses are commoner in women than in men. They tend to begin later and to last longer than arteriosclerotic mental disease. The onset is gradual and the course steadily progressive, leading eventually to profound mental deterioration.

Arteriosclerotic psychoses are commoner in men than in women. They may begin gradually or abruptly. The acute onset is marked by a confused or delirious state, with or without neurologic accompaniments. If this does not terminate fatally, it tends to subside, leaving as a rule a certain degree of mental deterioration, but occasionally leading to a good remission. When the onset is gradual the picture is similar in many ways to that of senile dementia, both disorders presenting signs of intellectual failure, frequently with emotional reactions and disturbed behavior. In arteriosclerotic psychoses, however, attacks of confusion may occur at any point, thereby producing a more stormy course with fluctuations in the severity of the mental disturbances.

The intellectual impairment is usually more pronounced in senile than in arteriosclerotic conditions. An exception to this is provided by the paranoid type of senile psychosis, in which the intellectual faculties may be relatively well preserved. However, outspoken and chronic paranoid syndromes are seldom noted in arteriosclerotic disorders. Severe hypochondriacal-like

symptoms are often an early feature of the latter, but are not ordinarily prominent in senile psychoses.

While gross neurologic changes are characteristic of arteriosclerotic psychoses, they often fail to occur in the earlier stages, and they may be completely lacking. Cardiac disturbances are common in arteriosclerotic patients. Other characteristic but less frequent symptoms are headache, dizziness, syncopal attacks, convulsive seizures and explosive laughter or weeping.

Sclerosis of the peripheral arteries occurs with equal frequency in the two psychoses. Hypertension is almost as common in senile as in arteriosclerotic patients, though the latter are more apt to show increased diastolic pressures.

Diagnostic problems involving other mental disorders are discussed. The commonest sources of error are provided by psychoses associated with a variety of toxic or somatic conditions, some of which may be particularly difficult to separate from arteriosclerotic mental disease. Many mistakes owe their origin largely to an uncritical attitude whereby one tends to forget that mental illnesses which are neither arteriosclerotic nor senile are common in later life.

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GERIATRICS CHAPTER IS ORGANIZED IN WATERBURY, CONNECTICUT

C. H. Neuswanger, M.D., and Jacques Gancher, M.D., have been named a special committee to formulate rules and regulations governing activities of the newly organized Waterbury Chapter of Geriatrics. They will submit a report at the next meeting of the chapter.

Officers of the chapter include: H. J. Stettbacher, M.D., president; O. J. Bizzozero, M.D., vice president; Arthur F. Sullivan, M.D., secretary; John H. Dillon, M.D., treasurer.

GERIATRIC RESEARCH LABORATORY IN BROOKLYN, NEW YORK

The Brooklyn Hebrew Home and Hospital for Aged, New York, announces that it has opened the city's first research laboratory devoted entirely to the study of geriatrics. This laboratory, which opened on January 1, was made possible by the Dora Brenner Research Fund.

THE FUTURE OF THE OLDER WORKER

Theodore Klumpp, M.D.

IN THE past, medical research has been largely preoccupied with the diseases of youth and childhood. Our chief endeavor was to learn to run the gauntlet of infections so that we might be able to raise a reasonable number of our children to adult life. We have not yet finished this job and we never will altogether—but mortality statistics show that we have made great advances for which our physicians, research workers, and public health officials, can feel justly proud.

But as a result of our success in conquering infections and some of the most deadly diseases of childhood, we now have an adult population that is larger in proportion than it has ever been before. It promises to grow even greater as the years roll by. This poses new public health problems, and hitherto neglected diseases assume compelling importance. The care of the aged must not be left to the old man with the long white beard and the scythe.

In a sense we are now standing where the stream divides. For generations we have followed the branch of infectious diseases. It has dwindled in size, but we have not yet really started the exploration of that other stream that flows in the direction of the chronic diseases of adult life and geriatrics. A whole new field of knowledge is waiting to be studied and mastered. We haven't even begun to think seriously about the sociological and public health aspects of old age and the chronic diseases. Neither medicine, nor industry, nor the State, has any carefully thought out program of what to do about the vast population of older persons that is rising in our midst. Only Dr. Townsend has a $50.00 every Friday plan which most other doctors feel is not the answer.

Our research has not scratched the surface of such problems as heart disease and coronary thrombosis, nephritis, arthritis, and cancer. We know more about the planet Mars than we know about the pathogensis of arteriosclerosis which is probably the least common denominator of most of the disabilities of the aging process.

At the present time we actually know pitifully little about the factors that influence the rate at which we go down hill. Among the few well established facts concerning longevity are these: Throughout the animal kingdom males are outlived by females. But here our knowledge ends and without

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an endocrinological, chemical, or physical reason to explain it, this information is not much help. We also know that obesity is prejudicial to long life. Of the individuals that have lived to a ripe old age, how many have you seen that are much overweight? Very few, I venture to say. The very old person is almost invariably lean and often very thin. But what else influences longevity? What diets, for instance, predispose to long living? Should we eat much meat or little? Is a vegetarian diet beneficial? Should we drink lots of milk, or Bulgarian bacillus milk, or lots of water instead? How about eggs? Dr. Leary, the distinguished Boston pathologist, believed that foods rich in cholesterol, such as eggs and cream, predispose to arteriosclerosis and should be avoided, but most pathologists disagree with him. How about exercise and physical work? Should we keep on exercising, or should we take a great lawyer's advice to the effect that when you feel the impulse to exercise coming over you, sit very quietly until it passes off, and then just keep on sitting? Or should we follow the adage that hard work never killed anyone?

What about fresh air, sunshine, vitamins, vacations, 8 hours of sleep, cold baths, sexual activity, alcohol, and tobacco? There are opinions on these questions; but from the standpoint of scientific proof, very few established facts. Some years ago, the late Raymond Pearl studied statistically the mortality records of smokers and non-smokers. He came to the conclusion that smoking had an adverse effect upon longevity. He may be right, but I am convinced that his studies did not prove the point. All he showed was that smokers do not live as long as non-smokers. And the reason I think he failed to prove it is this: As a statistical group there are other significant differences between smokers and non-smokers. The smoker is apt to be a more venturesome nervous type. He is the fellow who has to be doing something, who has to keep his hands occupied. He places a high valuation on sensual stimuli. The non-smoker is a fellow who successfully resists all the temptations to smoke that in our present-day society bombard him every day. If he says "no" to smoking he is likely to give the same answer to other things that might get him into trouble and shorten his life. As a group, the non-smoker must represent a less nervous, more placid, sterner, temptation-resisting, less venturesome type. It is not possible that these inherent differences in constitutional and nervous characteristics are factors that influence the life expectancy of these two groups, and may actually overshadow the effects of the smoking itself? One thing is certain, many smokers have lived to a ripe old age. This proves only that smoking is not incompatible with long life. But it does not prove that smoking does not shorten life, and in the end Raymond Pearl's conclusion may be correct though his data may have failed to prove it.

It is clearly apparent that as far as the cellular elements of our bodies are concerned we could, as a species, live many years longer. The cells of our body are capable of dividing and renewing themselves almost indefinitely,

even as the simple paramecium. Incidentally, barring accidents the paramecium possesses a kind of immortality in eternally dividing, growing, and dividing again. But man's complicated large mass of differentiated protoplasm requires an internal transportation system, his circulation, and this appears to be the unit that is the least common denominator of longevity, the limiting factor. It is this that will have to be made more enduring if we are to achieve an age of man that is substantially greater than we now enjoy. But this is in the last analysis a chemical problem. The aging of our blood vessels is accompanied by a loss of elastic fibers and a deposition of scar tissue and calcium salts. This is arteriosclerosis. But arteriosclerosis does not happen by spontaneous degeneration. There are subtle chemical changes mediated by changes in endocrine functions, which lead to the structural manifestations recognized as arteriosclerosis. We have learned to detect the obvious structural end result, but we know nothing (I think I am justified in using the absolute term) about the functional chemistry that leads to hardening of the arteries.

Despite our abysmal ignorance of the factors that influence long life, the life span, not content to wait for the facts and explanations to catch up with it, has been quietly increasing nevertheless. This is bringing a steady rise in the number of older individuals in our population. According to U. S. Census figures, and reliable estimates with respect to the future, the following represents the proportion of our population 45 and 65 years of age and over:

	45 Years and over	65 Years and over
1860	13.1%	2.7%
1880	16. %	3.4%
1900	17.8%	4.1%
1920	20.8%	4.7%
1940	26.5%	6.8%
1960	33.3%	10.0%
1980	40.3%	14.4%

The important facts to be derived from these data are that the weight of our population is shifting toward the older group and by 1980 two-fifths of our population will be over 45, and one-seventh over 65. At the same time the total number of our people is also increasing. It has been conservatively estimated that we will have not less than 150 million people by 1980. This means that by that time there will be not less than 60 million 45 years and over and over 21 million 65 and over. Think of it, 21 million persons 65 and over! These figures have special significance in the light of the all time employment record which has recently been announced by the President as 58 million gainfully employed. Which means that in less than 34 years we shall have more individuals over 45 years of age than the total number employed at the present time. We must also anticipate the continued

introduction of new labor saving devices perhaps on a scale never dreamed of before. The technologies recently developed, particularly in electronics and the upswing of interest in science generally should provide for that. If we do nothing about it and maintain present day working standards and conditions we shall have a potential labor surplus of over 30 million workers by 1980.

What to do about the aged? People are not happy when they are idle and this is particularly true of older individuals. Youth can loaf, content with opiate dreams of future achievements. But as we grow older the realities of life are more clearly seen and less easily denied and as we approach 50 and 60 we can no longer derive solace from the pipe dreams of future achievements. Similarly, the diversion of participation in sport and exercise are denied us. Age plays for real stakes not pastimes. It wants something to do and it must be real. And the most real thing we have to sustain us in this life of ours is useful work for which we are paid in money or its kind. Those who have seen, as I have, the compulsory retirement of faithful employees who have been working at their jobs for 20, 30, or 40 years to the boneyard by some blind compulsory retirement scheme, know what I am talking about. I believe I understand why so often they die shortly after retirement. Any biological organism that has been accustomed to a set routine for 40 or 50 years cannot suddenly be shaken from its orbit without untoward consequences. Anyone who has studied Cannon's ideas on homeostasis will recognize this.

Society has been quite illogical and inconsistent in its attitude toward the older worker. On the one hand it is apparent that we have no objection to electing and appointing older individuals to positions of the greatest responsibility in government, business, and the professions.

For instance, in the present Congress 32 Senators or 41.3 per cent are over 65 years of age, 12 or 15.3 per cent are 61 to 65 years of age, in other words, over 56 per cent are over 60. In the House of Representatives, 57 or 21.3 per cent are over 65 years of age, and 36 representatives or 13.4 per cent are 61 to 65 years of age. A study was made of top business executives as listed in Poor's Directory. Taking 500 consecutive names contained therein it was found that 143 or 28.6 per cent of those listed were over 65 years of age, 78 or 15.6 per cent were between 61 and 65 years inclusively. Here again, over 44 per cent are over 60 years of age. I am certain that a study of the leadership of the various professions would reveal the same large proportion of individuals in the older group.

And yet as far as the rank and file of workers is concerned, we have no objection to the imposition of blind and unselective compulsory retirement rules which automatically eliminate those in the ranks who have reached the same age regardless of their fitness, ability, and contribution to the group for which they labor. More precious than oil or fertile soil, than ore and minerals, than trees or an equable climate, are the human resources of a

country. All other things were here when Columbus came to America, but it took intelligent, industrious men to make our country what it is today. We may not fully realize it, but we cannot afford to waste the contributions of those who through years of experience have learned to do their jobs well and are willing and able to continue to do so faithfully. Training and experience can only be replaced by training and experience and when we retire a competent older worker his successor has the burden, inevitably, of supporting him as well as himself. Some individuals welcome retirement and the possibility of voluntary retirement on a pension should always remain open for these. However, if the premise is that all individuals over 65 or 70 are not worth their keep then least of all should we permit individuals above those ages to occupy the top and critical positions in our social structure. If we acknowledge, as is certainly true, that some are and some aren't fit and pulling their weight at those ages, then we should use our intelligence to devise standard methods of determining the fitness of all older workers. As the great physiologist, A. J. Carlson stated, "The physiologic age of the worker is not synonymous with his chronologic age, owing to the individual variables in heredity, mode of living, accidents and sequelae of disease." Likewise, "All age changes come on gradually" according to Carlson, and we know now that in some individuals changes characteristic of senescence begin even in childhood.

We choose and select when we hire, and I see no reason why we cannot do the same thing when we retire our workers. In 1930 on the basis of a compulsory retirement rule, the most distinguished neurological surgeon the world has yet known, Harvey Cushing, was retired from the Harvard faculty at a time when he was at the peak of his ability. He promptly accepted a full time academic appointment at Yale where he continued his outstanding contributions to medical science. What was Harvard's loss was Yale's gain, but for Dr. Cushing the retirement rule meant only the waste of time and inconvenience of pulling up stakes, moving, and finding a new home. When Dr. Milton J. Rosenau, Professor of Public Health, was retired from the same faculty he, too, moved to another University where the rules were not so blind. From a sociological standpoint the whole thing is ridiculous, and a reflection on the state of our intelligence in solving what ought to be a simple problem. Whenever society adopts a rule that eliminates the fit with the unfit, destroys the good with the bad, or punishes the innocent with the wicked, it is not a good rule. Society progresses by changing rules of this kind. In an imperfect society human beings are pushed around as a faceless mob. But social progress may be measured in the last analysis by the degree of skill and discrimination with which society solves the individual problems of its members.

The argument has been advanced that we must clear out the older workers to make room for the younger men so that their progress upward in an organization will not be unduly stymied. On the face of it this line of

reasoning appears to have some merit. But it is only another way of stating that there are more workers than there are jobs. During the war when there was a manpower shortage and no one was afraid that the old, the lame, the blind, and the halt were taking jobs away from younger and more able workers. At other times, similar arguments have been applied against the employment of women in business, government, and the professions. Certainly there is no arbitrary age at which older workers begin to repress the advancement of younger individuals. In a sense, every older individual higher on the ladder of advancement, whether he be 65 or 55 or 45 is holding a job that a younger individual aspires to and feels he can fill. That is always true and it will be just as true if we force everyone to retire at 50 or even 45 as we will have to do by 1980 if we do not find a more logical way of reducing the disparity between jobs and workers. We must not lose sight of the fact that someone must support those that we retire to idleness. The more workers we retire and particularly if we should lower the retirement ages, the greater will be the economic burden we will place on those who work. They will have to produce enough to support themselves and their families as well as the increased numbers of those who become emeritus workers. The whole problem is the adjustment of the number of workers to the number of jobs available. To attempt to strike a balance by eliminating all workers over a certain age is an unfair penalty on age and experience. In a refined and delicate way it is a perpetuation of the jungle law of the fang and claw where the leaders of the pack survive only until the younger beasts grow fierce enough to eliminate them. In modern civilization we are less violent but the end result is approximately the same.

One of the most important steps in solving this problem is the successive reduction in working hours for all workers rather than by reducing more and more older workers to a state of parasitism. I venture to say that we will see a thirty-hour week in industry within twenty years.

Another view that is widely held is that older individuals become overconservative, and to make progress we must eliminate these obstacles in the path of progress. Here again there are such wide variations in human reaction patterns that I do not see how one can logically draw generalizations that will fit individual cases. Certainly Bernard Baruch, 76 years of age, and Arturo Toscanini, at 80 are no obstacles in the path of progress, and neither Senator Pepper nor Henry Wallace have become more conservative as they have grown older. As far as the majority of jobs now subject to compulsory retirement is concerned, it makes no difference at all whether an individual grows more or less conservative. If individuals in key positions become too conservative with age or even too reckless as they sometimes do, to meet the best interests of the organization, a retirement board operating on a selective basis can function to correct this development, as well as any others that may arise.

During the past decade, industry and our Federal and State Govern-

ments have made great advances in provisions for the care of the aged. What we have accomplished is only a start and much more remains to be done. I am hopeful that the major burden of this can be carried by the extension of voluntary retirement plans in which the employer and the employee share the cost. Not only must more realistic retirement benefits be provided, but such plans must be more widely adopted by employers generally. The problem of transferring retirement benefits without loss when an employee moves from one job to another remains to be solved. Under present day practices a worker who changes from one job to another to advance himself or for any other reason, is penalized by loss of the portion of his retirement accumulation which the employer contributes. The fear of losing such retirement benefits stands as a formidable obstacle to individual advancement in industry. For those not covered by adequate voluntary retirement programs, the present token social security benefits must be increased, and old age assistance on a lower scale provided for those who, for one reason or another, have failed to earn their share of the retirement burden.

While social advances in the direction I have indicated will cost money, there is no contribution we can make to human welfare and happiness that is of greater fundamental importance. Compared with the tax burden of war and armaments, which human ingenuity should be capable of eliminating, it is negligible. Even this tax burden can be lessened by permitting capable and willing older workers to continue working, and finding more places in industry and government for those partially disabled.

As we progress to a more perfect civilization, the goal of which is nothing more than a greater measure of happiness for all mankind, there is much that remains to be done for the aged. The following general principles will, in my opinion, serve as a guide toward this end:

1. Since physiological age is not synonymous with chronological age, compulsory retirement on a calendar age basis should be abandoned.
2. Since hiring is selective and based on fitness to do a given job, retirement should likewise be selective and based on unfitness. If we can do one there is no reason why we can not do the other.
3. Compulsory retirement should be based on the recommendation of a retirement board composed of medical and psychiatric members as well as administrative officials.
4. In accord with Carlson's idea, if wage in proportion to performance is recognized as a fundamental principle, the older worker should taper off in industry, just as the young apprentice gradually works himself up in skill, performance and remuneration. In other words, opportunities for down-grading in position and salary should be offered. If this becomes a general practice, the aura of foolish pride which now stands in the way of this would soon disappear. The older worker can choose retirement if he does not like the idea.
5. When the aged have work to do they are less of a burden, financially,

socially, and spiritually to the folks at home. Other things being equal, the strong and fit must learn to exercise a greater measure of patience, tolerance and kindness toward them.

6. Institutions for the aged must be changed from asylums to modern institutions where every convenience and scientific development is available for their physical, mental and spiritual comfort. The importance of occupational therapy must not be forgotten. Useless work such as basket weaving is not the answer. Under ideal conditions each institutionalized person will have a job tailored to fit his ability and aptitude and for which he will be paid in proportion to his production.

This paper has only one purpose: To emphasize the growing importance of the problem of the aged. It is a plea for the devotion of more thought, more research, and more funds to improve the lot of the largest and most neglected group of unfortunates in our society. It is from those interested in public health and welfare that we may expect leadership in this endeavor.

Old age is not a disease, but the disabilities arising from it are. As we overcome these we not only postpone old age but we defeat its sorrow and suffering. Death has its final victory but it can come in peace.

STUDY OF ARTERIOSCLEROSIS

The constantly increasing importance of arteriosclerosis as a leading cause of death and as a prevalent source of prolonged physical and mental disability makes the early institution of effective measures for the control of this general health hazard urgent.

Plans have been completed to organize the American Society for the Study of Arteriosclerosis. Clinicians and experimentalists interested in a concerted intensified investigation of all phases of this disease are invited to join this organization and are urged to contact as soon as possible Dr. O. J. Pollak, Wilmington General Hospital, Chestnut and Broom Streets, Wilmington 14, Delaware.

The constituting meeting has been called for Monday, June 9th, 1947, at 9:00 a. m. at the Hotel Strand, Pennsylvania Avenue and Boardwalk, Atlantic City, N. J.

Members of the Organizing Committee are:

Clinicians—G. R. Herrmann, Galveston; R. A. Katz, New Orleans; W. B. Kountz, St. Louis; J. B. Wolffe, Philadelphia.

Experimentalists—G. L. Duff, Montreal; H. Goldblatt, Los Angeles; W. C. Hueper, New York; I. H. Page, Cleveland.

NITROGEN BALANCE STUDIES IN ELDERLY PEOPLE

William B. Kountz, M.D., Lilli Hofstatter, and Philip Ackermann, PH.D.

MEDICAL literature indicates that 1 gram of protein per kilogram of body weight is adequate for the diet of the normal individual. The adoption of this amount of protein was based upon a great number of nitrogen balance studies performed by different workers. According to Benedict et al,[1] Leitch,[2] Sherman et al,[3] Bricker et al[4] and others, a protein allowance of 1 gram per kilogram of body weight per day is about 50 per cent higher than the amount of protein needed for the maintenance of nitrogen equilibrium in the normal adult on a mixed diet.

The optimal protein requirement is believed to be higher. Studies on the Army, Navy, and Aviation personnel[5, 6, 7] showed that higher protein diets increased their efficiency. Such could not be confirmed by Darling[8] who found no relation of the amount of protein in the diet to physical vigor in a short period study of eight young men.

In the literature one finds very little attempt to investigate the maintenance requirement of protein of older people. Low protein diets have been suggested by some observers[9, 10] because of the individual's diminished power of dealing with excess food. It is believed that surplus protein is less readily absorbed and catabolized, that there is an increased susceptibility to excessive putrefaction and also there is no further need for tissue building material for growth and the repair quota is at a minimum.

Protein restriction in the diet of the elderly has also been thought to be a necessary precaution against promotion of renal injury or damage to blood vessels.[11] The work of Newburgh,[12] however, indicated that protein is not necessarily harmful, that the degree of renal injury depends mainly on the character of the protein and less on the concentration or length of

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feeding. Kidney damage in liver fed rats was severe and occurred after a short period of time in contrast to rats who were fed with casein. Newburgh considers diets containing 12 to 18 per cent of protein as harmless. McCay's studies[13] on life span in experiments on rats were suggestive that a more liberal protein diet may be permissible in older people. He found no significant change when the protein level of the diet was maintained at from 8 to 30 per cent.

The present thought in the literature is that the protein in the diet of the elderly should be more liberal. The amount generally agreed upon is 1 gram of protein per kilogram of body weight per day.[11, 14, 15] These authors point to the importance of protein in the protection of the liver, its effect upon the specific dynamic action, the role of protein in the resistance against infection[16] and the sense of well being which is also attributed to the protein in the diet.

Sporadic cases have been reported in the literature of nitrogen balance studies made on elderly individuals. None of these studies would indicate whether or not a protein allowance of 1 gram per kilogram of body weight per day would actually meet the protein requirement in the aged and maintain these individuals in nitrogen equilibrium. Striek[17] reported that he found no increased nitrogen excretion after heavy labor in a 70 year old man who had been living for years on a diet containing 30 grams of protein a day. Fenger[18] observed a woman over a period of 15 years who had been maintained in nitrogen balance and a good state of nutrition on a protein intake of 1.9 to 2.0 grams per kilogram of body weight per day. She took no meat during this entire time and was chiefly on liquid food. Koch[19] found five elderly men in nitrogen equilibrium who had received 1.6 grams of protein per kilogram of body weight per day.

Clinical studies dealing with older individuals would seem to emphasize that nutrition is of paramount importance in the control of their health. The wasting of their body tissues would suggest that their protein metabolism may be disturbed. Nitrogen balance studies made on elderly individuals and reported in the literature do not give us adequate information in this regard because the studied subjects were either too few, the diet offered was not a normal one or the protein intakes were 50 to 100 per cent higher than the amount recommended at present.

We, therefore, would like to introduce a study of nitrogen balance in a larger number of elderly people under complete dietary control.

MATERIAL AND METHODS

Nitrogen balance studies were made on 27 elderly people, 13 women and 14 men. Their clinical diagnoses were the following: 14 cases of arteriosclerosis, 4 mental defectives, 3 cerebral accidents some years previously, 1 hypertension, 1 post-coronary, 1 female with congenital syphilis, 2 patients with degenerative muscular disease and 1 degenerative arthritis with arterio-

sclerosis. None of the patients used in our results were bedfast or acutely ill during any of the time of the test period or the preparatory weeks before the actual observations were begun.

In order to permit dietary adjustment, three weeks previous to the beginning of the test period the patients were put on the same diet under which the nitrogen balance was to be tested. The diet averaged from 2020 to 2040 calories per day, 17 to 18 per cent protein, 34 to 36 per cent fat and 46 to 48 per cent carbohydrates. The protein was supplied mainly in form of beef or veal, milk, cheese, cottage cheese and eggs. The food was weighed to the patients and the rejected amounts were subtracted from the offered food. Food substances such as meat, all desserts and other foods inconstant in their nitrogen contents, were analyzed daily for nitrogen during the test period. Other substances such as cereal, canned vegetables, milk, etc., which gave the same nitrogen values on at least six occasions, were thus considered to be constant in their nitrogen content and were not retested each time they were offered to the patient. Tables of Bowes and Church[20] were consulted for the carbohydrate and fat content of the foods used.

The menus were rather monotonous but were simple in their preparation. Vegetables were steamed in water, meats were ground and broiled without fat and desserts were restricted to custard, puddings, ice cream or stewed fruits. Weekly repetition of whole menus and the simple type of preparation of the foods was used in order to reduce the number of necessary chemical analyses. This type of feeding was also of advantage in decreasing the variations of actual food intakes from day to day, as the patients, who had developed likes and dislikes for certain food substances, chose from a limited menu.

Chemical Procedure: Twenty-four hour urine specimens and stools were collected during the same period and were analyzed for the total nitrogen content by the Kjeldahl method.[21] To make sure that the volume was complete, the urines were first examined for total creatinine, using the Folin-Wu method,[22] and specimens were used only when the total creatinine content corresponded with that of previous examinations.[23] The stool specimens of each patient were collected for the day; sufficient water was added to insure adequate mixing and the mixture was thoroughly stirred and shaken. From this an adequate amount of the stool was taken for the nitrogen determination. The total nitrogen excretion in stools and urines for each day has been averaged and tabulated.

The results have been divided into 3 groups according to their nitrogen balances. (See Table 1) Nitrogen balances ranging between —.5 and +.5 were used as the upper and lower limits of nitrogen equilibrium. Nitrogen balances below or above these values were considered to be negative and positive, respectively. The data of each patient in Table 1 are average values for three days. The ingested food is the offered food minus the rejected food.

RESULTS

TABLE 1.
NITROGEN BALANCE STUDIES

Patients	Age	Ingested Food in Grams: Protein	Fat	CHO	Calories	Nitro. Intake in Grams	Protein per Kgm. Weight in Grams	Nitrogen Output in Grams: Urine	Feces	Nitrogen Balance in Grams	Creatinine Urine in Grams
GROUP 1											
B.A.	50	84.2	71	190	1738	13.4	1.2	11.7	2.6	— .9	1.03
A.H.	71	61.7	58	236	1715	9.9	1.1	11.2	2.1	—3.4	.98
F.E.	46	61	64	223	1712	9.9	1.0	8.9	3.2	—2.2	1.13
H.J.	75	77.6	68	207	1751	12.4	1.4	11.0	2.9	—1.5	.82
S.P.	63	90.9	87	238	2097	14.5	1.2	14.4	1.6	—1.5	1.19
M.R.	80	48.3	51	213	1506	7.7	1.0	9.0	1.4	—2.7	.65
S.M.	64	59.1	59	213	1614	9.5	1.3	10.2	1.2	—1.9	.89
S.M.	57	49.4	56	212	1548	7.9	1.4	9.7	1.1	—2.9	.70
R.A.	86	41.1	47	134	1121	6.6	1.0	6.1	1.3	— .8	.44
S.T.	59	86.2	82	222	1974	13.8	1.1	14.8	1.9	—2.9	1.22
Y.C.	55	79.5	82	210	1892	12.7	1.8	11.4	2.0	— .7	.83
Average	64	67.1	66	209	1697	10.8	1.2	10.8	1.9	—1.9	.89
% of Total Calories		15.8	34.8	49.4	100						
GROUP 2											
F.F.	41	74.3	71	236	1880	11.9	1.3	10.7	1.6	— .4	.85
C.A.	81	89.9	77	241	2017	14.4	1.5	12.4	1.7	+ .3	.96
F.E.	54	88.1	76	223	1926	14.1	1.4	12.0	1.6	+ .5	1.17
F.C.	62	71.3	79	219	1867	11.4	1.0	10.8	1.0	— .4	.77
J.A.	79	60.6	65	215	1688	9.7	1.2	7.9	2.2	— .4	.67
L.C.	51	92.3	77	226	1967	14.8	1.4	12.1	2.3	+ .4	1.45
W.W.	85	54.8	68	168	1506	8.8	1.8	6.5	1.9	+ .4	.35
G.B.	84	77.9	77	235	1990	12.4	1.9	10.2	2.1	+ .1	.56
Average	67	76.1	74	220	1849	12.2	1.4	10.3	1.8	+ .1	.85
% of Total Calories		16.5	35.8	47.7	100						
GROUP 3											
D.G.	56	75.2	71	228	1855	12	1.3	10.0	1.2	+ .8	1.26
F.E.	79	72.4	69	255	1929	11.6	1.4	8.5	2.4	+ .7	.84
W.J.	68	91.3	87	236	2090	14.6	1.6	10.8	2.3	+1.5	.96
S.J.	74	89.9	83	234	2044	14.3	1.5	10.2	1.7	+2.4	.77
M.H.	70	91.2	72	240	1977	14.6	1.4	8.6	1.4	+4.6	.89
O.M.	69	77.4	65	234	1833	12.3	1.3	10.6	.8	+ .9	.95
P.B.	57	84.4	72	214	1836	13.5	1.4	9.6	.8	+3.3	.87
S.M.	51	93.3	80	235	2033	14.9	1.4	11.7	1.8	+1.4	1.13
Average	65	84.4	75	234	1950	13.5	1.4	10.0	1.5	+2.0	.96
% of Total Calories		17.3	34.6	48.1	100						

The ingested food, or the amount of food eaten, was frequently lowest in the individuals who showed a negative nitrogen balance. This, however, was not always true. In none of the patients was the protein intake per kilogram of body weight below 1 gram. The average was 1.2 grams for the group in negative nitrogen balance and 1.4 grams for the group in nitrogen equilibrium and for the group in positive nitrogen balance.

TABLE 2.
THE INCIDENCE OF NITROGEN BALANCE UNDER VARIOUS PROTEIN INTAKES

Protein Ingested per Kgm. Body Weight in Grams	Types of Nitrogen Balance — Negative No. of Pts.	%	Equilibrium No. of Pts.	%	Positive No. of Pts.	%	Total No. of Patients in Each Group
1.0—1.2	5	83.5	1	16.5	0	0	6
1.2—1.4	3	42.8	2	28.6	2	28.6	7
1.4—1.6	2	20.0	3	30.0	5	50.0	10
1.6—1.8					1		1
1.8—2.0	1		2				3
Total	11	40.8	8	29.6	8	29.6	27

Correlation of the protein intake per kilogram of body weight with the nitrogen balances of these patients was interesting. (See Table 2) Of the 27 patients 40.8 per cent, or 11 patients, were in negative nitrogen balance, or tended to excrete more nitrogen than they took in. Eight were in equilibrium, while 8 tended to retain nitrogen or were in a positive nitrogen balance. From Table 2 it can be seen that of the 6 patients with a protein intake of 1 to 1.2 grams, 5, or 83.5 per cent, were in negative nitrogen balance and only 1 in nitrogen equilibrium. With a protein intake of 1.2 to 1.4 grams, 3 out of 7 patients were in negative balance, while 2 out of 10 were in negative balance with a protein intake of 1.4 to 1.6 grams. One out of 3 shows a negative nitrogen balance with a diet containing 1.8 to 2.0 grams of protein.

TABLE 3.
THE INCIDENCE OF NITROGEN BALANCE UNDER VARIOUS AMOUNTS OF PROTEIN ABSORPTION

Protein Absorbed per Kgm. Body Weight in Grams	Types of Nitrogen Balance — Negative No. of Pts.	Equilibrium No. of Pts.	Positive No. of Pts.	Total No. of Patients in Each Group
.6— .8	3	..	..	3
.8—1.0	1	2	..	3
1.0—1.2	4	2	2	8
1.2—1.4	2	3	6	11
1.4—1.6	..	1	..	1
1.6—1.8	1	..	..	1

We attempted in Table 3 to evaluate the absorbed protein. We did this by subtracting the nitrogen of the stool from the total nitrogen ingested. We, of course, neglected the bacterial nitrogen, an error we could not avoid.

TABLE 4.
COMPARISON OF FOODS OFFERED AND REJECTED

Patients	Food Offered in Grams				Food Rejected in Grams				Food Rejected Expressed in % of Offered Food			
	Protein	Fat	CHO	Calories	Protein	Fat	CHO	Calories	Protein	Fat	CHO	Calories
Group 1.												
B.A.	85	72	221	1866	.8	0	31	129				
A.H.	91.5	74	274	2124	29.8	15	38	408				
F.E.	79.4	71	241	1922	18.4	7	18	210				
H.J.	83.4	69	224	1850	5.8	1	17	97				
S.P.	90.9	87	238	2097	0	0	0	0				
M.R.	96.9	82	245	2099	48.6	31	31	595				
S.M.	90	72	258	2038	30.9	13	46	424				
R.A.	87.8	88	271	2224	46.7	41	137	1103				
S.M.	88.3	89	272	2244	38.9	33	60	696				
S.C.	86.2	82	222	1974	0	0	0	0				
Y.C.	93	85	225	2034	13.5	3	15	142				
Average in Grams	88.4	79	245	2043	21.2	13	36	346				
% of Total Calories	17.2	34.7	48.1	100					24%	16.1%	14.5%	16.8%
Group 2.												
F.F.	82	72	245	1958	7.7	2	9	77				
C.A.	89.9	77	241	2016	0	0	0	0				
F.E.	88.1	76	223	1926	0	0	0	0				
F.C.	93.7	92	237	2144	22.4	13	18	277				
J.A.	88.1	82	223	1986	27.5	17	9	298				
L.C.	92.8	81	237	2046	.5	4	11	80				
W.W.	92.8	81	237	2046	38	25	69	657				
G.B.	93.5	91	243	2167	15.6	15	8	227				
Average in Grams	90.1	81	236	2036	13.9	9	15	202				
% of Total Calories	17.8	36.1	46.5	100					15.4%	11.5%	6.5%	9.9%
Group 3.												
D.G.	85.2	77	239	1991	10	6	11	136				
F.E.	90.3	78	265	2118	17.7	9	10	189				
W.J.	91.3	87	236	2090	0	0	0	0				
S.J.	90.5	84	243	2089	1.0	0	10	46				
M.H.	94.2	73	245	2009	3.0	0	5	32				
O.M.	89.2	69	254	1997	11.8	4	8	118				
P.B.	85.4	72	216	1849	1.0	0	2	13				
S.M.	93.3	80	235	2033	0	0	0	0				
Average in Grams	90	77	242	2022	5.6	2	6	67				
% of Total Calories	17.8	34.4	47.8	100					6.2%	3.1%	2.3%	3.3%

In none of our patients did we find the total nitrogen absorbed to be less than .6 grams of protein per kilogram of body weight, but all the patients who had absorbed only .6 to .8 grams per kilogram of body weight were in negative nitrogen balance.

In order to investigate whether food habits play a part in the frequent occurrence of negative nitrogen balances, we tabulated the offered and rejected food substances for each group of patients. (See Table 4) The food offered is approximately the same in calories as well as percentage distribution of the various food stuffs for each group. (See left hand columns in Table 4) The rejected food in grams of each individual is averaged for the observation period. Although some of the patients in negative nitrogen balance have left no food on their trays, the average rejection of the group is larger than of the groups of patients in nitrogen equilibrium or in positive nitrogen balance. The right hand columns indicate the average percentage of rejected portions of offered food for each group.

It may be noted that 24 per cent of offered protein was rejected by the patients in negative balance. This compares with a rejection of only 15 per cent and 6 per cent of protein by patients in nitrogen equilibrium and in positive nitrogen balance.

DISCUSSION

As shown in Table 2 a protein intake of 1.0 to 1.2 grams per kilogram of body weight was found insufficient to maintain nitrogen balance in 5 out of 6 elderly subjects. The percentage is 83.5. When the protein intake was increased from 1.2 to 1.4 grams per kilogram of body weight, 3 out of 7 people (42.8 per cent), 1.4 to 1.6 grams per kilogram of body weight, 2 out of 10 (20 per cent) were found to be in negative nitrogen balance. These findings would suggest that the protein requirement of the aged is higher than usually assumed and certainly is higher than the accepted standard allowance of 1 gram per kilogram of body weight. Since none of these patients had any disease which would contribute to an increased protein use[24] during the period of our observation, it must be assumed that some older people need more protein than do others. It is possible that different factors are responsible for these findings. Faulty food habits in individuals who do not eat adequate amounts of protein could be a cause. A second possibility might be the failure of absorption from the intestinal tract, whereas a third could be related to metabolic changes in older people.

Faulty Food Habits: All of our patients had been offered about the same diets. (See Table 4) The average total food intake, however, was less in the group of patients with negative nitrogen balance as compared with those in equilibrium and in positive balance. (See Table 1) One may also note that the amount of ingested protein was lowest in the former group (Group 1) as compared to that in the later two groups (Groups 2 and 3). This indicates that the food rejected by those in negative balance consisted to a large extent of protein, 24 per cent, and to a relatively large extent of

fats and carbohydrates, which were 16 and 14 per cent, respectively. (See Table 4, right hand column) These are the patients with a poor appetite and a dislike for meat, yet none of the individuals in negative balance had a protein intake of less than 1 gram per kilogram of body weight. The average for the group was 1.2 grams. (See Table 1) A simultaneous reduction of carbohydrates and fats, which are protein sparers,[9] may have contributed to increase the demand for protein. It is possible under such circumstances that the quantity of 1 to 1.2 grams of protein per kilogram of body weight became insufficient to maintain nitrogen equilibrium. This may have been the case in some of the patients, but such an explanation could not hold true for those in negative nitrogen balance whose protein, carbohydrate and fat intake was seemingly adequate. (See Table 1).

Failure of Protein Absorption: According to DuBois[25] in the normal individual ingested protein substances are completely absorbed from the intestinal tract. One finds, according to him, about 1 gram of nitrogen in the stools per day which is derived from debris of the intestinal wall, secretions, bodies of bacteria, and the like. Under certain circumstances, such as diarrhea, absorption of protein becomes incomplete and the amount of nitrogen in the stool is increased. Many of our patients showed a moderate increase in the amount of nitrogen in their stools, excreting more than 2.0 to 3.0 grams per day. (See Table 1) This suggests an incomplete absorption of the protein foods in some older people. It was found in patients of all three groups, regardless of the nitrogen balance. It was, however, more pronounced in those who were in a negative nitrogen balance. The nitrogen lost in the stool was too small to account for the obtained negative nitrogen balances. It may have contributed to the protein deficit especially in those who rejected considerable amounts of protein food.

In Table 3 we have charted the calculated absorbed protein per kilogram of body weight in relation to the incidence of the nitrogen balance. This Table is to be compared with Table 2, where the protein intake, without considering absorption, was correlated with the nitrogen balances. The absorbed protein per kilogram of body weight was .6 to .8 grams (none of the individuals studied had less absorbed) in three patients whose protein intake was 1 to 1.2 grams. All these people were found to be in negative nitrogen balance. In some patients nitrogen equilibrium was possible if the absorbed protein was .8 to 1.0 grams, while others were still in negative nitrogen balance with a protein absorption of 1.2 to 1.4 grams, amounts considered generally as very liberal. We believe, therefore, that insufficient protein intake plus insufficient absorption may play a role in some of the elderly individuals with a disturbed nitrogen balance but these factors cannot be considered the only explanation.

Metabolic Factors: Neither the failure of protein absorption nor faulty food habits would seem to be the explanation for the occurrence of negative nitrogen balance in a number of elderly individuals on diets, considered

adequate or even liberal in protein. One must assume, therefore, that some changes in the aged body must have taken place which are responsible for the increase of their protein metabolism. The nature of such changes is not known at the present time.

With increasing protein intake we found an increasing number of patients in positive nitrogen balance. A transitory lag of nitrogen excretion, not exceeding four days, occurs in the normal adult, whenever a higher protein diet follows a lower one and lasts until the metabolism has adjusted to the higher protein level.[9] Our patients had been given a three week's adjustment period from their so-called normal hospital diet, calculated on the basis of 1 gram of protein per kilogram of body weight per day, to the test diet which was about 20 grams higher in protein. It is possible that in the aged such an adjustment is greatly prolonged.

Further studies of the same patients on higher protein diets, which we shall report in another paper, have shown that their positive nitrogen balances could be kept up for months. A persistent positive nitrogen balance is not unusual in protein depleted individuals throughout the recovery period when sufficient protein is ingested.[9, 26] On so-called balanced diets, the majority of patients was found to be in negative nitrogen balance. This small, but continual nitrogen loss whether caused by faulty food habits, failure of absorption or metabolic changes of the aged must, by necessity, lead to a protein depletion of the body.

The foregoing study indicates that the protein requirement in the aged individual is more than 1 gram per kilogram of body weight per day, the amount usually assumed. Our group of patients was not large enough to estimate the actual protein requirement for the aged, but it seems that the protein in the diet should be substantially increased for the upkeep of proper nutrition and to safeguard the elderly patient from nitrogen loss.

SUMMARY

1. Nitrogen balance studies were carried out on 27 elderly patients. Each patient was offered a diet of about 2000 calories per day in the form of 17 to 18 per cent protein, 34 to 36 per cent fat and 47 to 48 per cent carbohydrates.
2. In the group of 27 patients we found 11 (40.8 per cent) in negative nitrogen balance. Eight patients were in nitrogen equilibrium and eight were in positive nitrogen balance. We also showed that a protein intake of 1 to 1.2 grams per kilogram of body weight per day, an amount considered liberal for this age group, was insufficient to maintain nitrogen equilibrium in 5 out of 6 patients, or 83.5 per cent. Even larger amounts, such as 1.4 to 1.6 and 1.8 to 2.0 grams of protein per kilogram of body weight per day, were not adequate, in many cases, to maintain the nitrogen balance of these people.
3. Although at the present time we are unable to account fully for the frequent occurrence of negative nitrogen balances, we believe that some of

the factors involved are: first, poor food habits; second, incomplete absorption, and third, metabolic changes of the aged.

4. Many individuals with a higher protein intake were found to be in positive nitrogen balance. The explanation for this could be an unusually slow adjustment from the previous lower protein diet to the higher one. A second explanation could be that previous protein depletion is followed by a period of protein accumulation in the body. Further investigations suggest the likelihood that such may be the process.

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COUNSELLING THE AGED

Clare deGruchy

THE FUTURE is the concern of the old as well as the young. Youth and age each has a contribution to make if we are to develop a higher concept of a democratic mode of life.

Yet today society is so organized that there is no place where the old can function fully. The result is that they have been pushed out of life and are forced to live in a past that has already become obsolete. Of necessity they grow more and more out of harmony with a present they do not understand and a future of which they are fearful, and thus act as an obstructive influence on the rest of society.

The old view that mental deterioration is the inevitable concomitant of aging has been proved by science to be fallacious. The normal mind can learn at any age, can re-claim life and contribute to society by gaining a realistic view of present-day conditions, and can resume a functioning that is an aid to progress.

The Old Age Counselling Center of San Francisco, founded by the late Dr. Lillien J. Martin and directed by her until her death in 1943, at the age of 92, was the pioneer effort to make practical a psychological-social education that would enable the client to create for himself intelligent participation in group living. The aim of old age counselling is to remove the frustration that society has hitherto imposed on the aging person; to help him release those potentialities latent in every human being, so that he may again play his part in life rather than remain a parasite to the end of his days.

To accomplish this the old age counsellor must consider his clients as individuals. To attempt to improve an unhappy condition through a change of environment without changing the individual who is to function in it generally results in failure.

Today old age counselling has passed the point of experiment. It has a well developed technique, the results of which have been tested at every point to prove their value for practical living. What has come to be known as the Martin method of old age counselling begins with a subjective study of the individual client: What manner of person is he? How does he look upon life? How does he see himself in relation to it? What role would he like to play in society?

When the client fully comprehends this self-revelation with respect to

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MRS. CLARE DEGRUCHY became associated with Dr. Lillien J. Martin, consulting psychologist, San Francisco, in 1919; Assistant, Old Age Counselling Center, San Francisco, 1929 to 1943; Director, Old Age Counselling Center since 1943 at the death of Dr. Martin; Co-author, *Salvaging Old Age, Sweeping the Cobwebs;* Author, *Creative Old Age.*

his past, present, and future life, he is re-introduced into group living to learn its present-day requirements, in order that he may participate in it as best suits his needs and capacities. He goes forth as a "self-starter in the right direction" to gain the place in the community that has so long been denied him—by self-survival through work, if that be necessary, or by self-fulfillment and exercise of his right to contribute to the welfare of all.

The four to five visits of an hour each, undeviatingly carried through with every client individually, are spaced a week apart, with sustained "home work" between visits. The first step in the subjective study of the client is to observe his general appearance, because such observation gives an idea of how he will impress others in any group he may attempt to enter.

After his name, age, and residence are noted, he is asked what brought him to the Old Age Counselling Center. Clients know quite definitely for what purpose they have come, and lose no time in stating their problems.

All physical disabilities, whether temporary or permanent, are given full consideration. No medical, neurological, or psychiatric advice is ever given in the Center. A complete report on the client's physical condition must be gained before any program for his future functioning can be made, for whatever plans are made with him must include recognition of his physical limitations and his medical instructions, so that he may carry on without strain.

The first visit is divided into three periods, allowing about twenty minutes for each. First is the narration of the life history, not through any formal questionnaire, but by asking the client to begin with his earliest memories and relate his life consecutively up to the present. We learn from this how his personality took shape, developed, and has been sustained. The reaction-pattern is prophetic. It has become automatic and if sustained one can forecast the results it evokes.

The second twenty-minute period of this first visit is used for giving a brief mental test of ten questions, eight of which are taken from the Stanford Revision of the Binet-Simon Intelligence Tests at 8-10 year level. The other two are Fernald's Ethics Test and an adaptation of a German test to evaluate the degree of suggestibility present. There is never any scoring of these tests, for the purpose is to learn qualitatively how much mental deterioration has been allowed to take place.

The final twenty-minute period is utilized by the counsellor to analyze with the client the results obtained through observation, the life history, and the mental tests. Through discussion of these, the handicaps under which he is functioning are agreed upon, and he is now prepared to consider his first "home work." This consists of a chart on which is marked the maximum of three points for daily practice, which he is to fill in and bring with him when he returns a week hence—such points as: Did I work toward improving my reaction-pattern today? Did I take my memory exercises today?

On his second visit the chart is discussed with him. But the main ob-

jective of this visit is to consider his daily use of time. It must be remembered that our aim in this respect is to have the daily life of every consulter divided among periods of work, recreation, and rest, according to his individual needs and conditions of living. The activities of each daily program should be planned so that each day promotes self-development, adequate rest, and some amusement that acts as true recreation to renew mental and physical functioning. For this purpose the client is given Chart II, which is marked off daily, hour by hour, for a week, and is asked to fill it in each evening and to bring it back on his following visit. Invariably the Daily Program record shows the counsellor if the client is leading a purposeful life or merely marking time.

The third visit to the Center is made for the purpose of discussing the client's income and its distribution. For this the Budget Chart is used on which are listed general expenditures for the necessities of life and also such items as amusements, gifts, services, savings, etc. The proportional amounts used for given purposes indicate clearly on which the client places emphasis. Beyond this, the counsellor may discuss several pertinent topics with the client, such as the value of a balanced budget, the influence of poverty and its lessons, and the obligations of wealth.

On the following visit, the fourth, the client comes for the purpose of establishing his future functioning as a good individual and a good citizen. His self-chosen goal becomes the motivation for his future life-plan. Sometimes, even after the full subjective study has been made, it is difficult for the client to express the nature of his goal. In such cases an effective question may be asked: "What would you do if you had your life to live over again?" His spontaneous answer is often a good guide to his emotional preference, which we might not learn otherwise.

Up to this point we have dealt with the client from the purely subjective point of view. On his fifth, and generally his final visit, we introduce him to a world outside himself. It is now the time to consider his participation in community life, but not before he understands the value of such participation. In general, old people have a very limited view of current world affairs. They must learn to think objectively not only of their own desires, but also of the needs, welfare, and progress of society as a whole.

The client's tendency will be to consider first what part he will play in a given project, rather than its significance to society. If allowed to become a participator in a work or recreation group without preparedness, he will tend to bring to it his outmoded ideas and practices, his obsolete beliefs, manners, and customs. He will not learn that participation must be *earned* and that cooperation implies new learning.

On the other hand, it is his right to express his views, which should be countered when necessary through discussion. To force our own views on him is worse than leaving him alone.

Our experience during the war years demonstrates that many old peo-

ple can be gainfully employed and are worthy of their hire. This compulsory salvage of the abilities of old men and women seems to offer a fitting occasion to consider the place of old age in the future, which we believe must be different from and better than the past. From the point of view of law and democracy, there is no right given to any group to set a limit, by means of counted years alone, to the individual's supporting himself and continuing his responsible part as a citizen.

In peacetime the majority of older people seeking jobs are met with sustained discouragement, because of the belief that the number of jobs is fixed and inelastic, that if the old people are allowed to retain their work or to become re-employed, the young will be left jobless. This is, of course, entirely inconsistent with the industrial expansion program of these postwar years. But one thing is apparent: the world can no longer afford to deny a chance for useful activity to a qualified worker at an arbitrarily fixed age. A constantly shrinking proportion of workers cannot continue to carry on their backs a constantly swelling load of consumers in enforced idleness. The limit of endurance has been reached.

In the days when the Old Age Counselling Center held clinics in business houses for employees whose work was falling below standard, three striking findings resulted. First, that slowness, lack of cooperation and resistance to change were found in workers of all ages. Second, that these characteristics generally had specific causes—such as home worries, hampering habits, unwholesome ideas and emotions, and similar situations other than old age alone. Third, that the causes creating maladjustment could be remedied in the old as in the young, by attacking them at their sources.

When the cause has been removed—as in the case of fear of dismissal —not infrequently the older worker has proved more valuable than ever before, and far from being ripe for retirement has responded with enthusiasm to increased responsibility. Even workers long unemployed, or persons who had never before worked away from the home, when rehabilitated in the Center proved that they could become excellent workers without entering into competition with the young.

But the results of the liberation of the spirit of old age from the shackles generally imposed on it in our times are even more important than the financial or economic results. Old people, whether rich or poor, can reclaim life, and through renewed self-esteem, courage, and re-education can put the treasures of their experience at the service of their fellow men and become assets rather than liabilities to the world. Society needs the fresh enthusiasms, the forward look, the vitality and resourcefulness of youth. But it needs also the kindly wisdom, the broad outlook, the mellow experience, and the tolerant impartiality of old people who have not declined into a bitter winter but have matured into a golden Indian summer of life. What we need now is faith that the old can be reborn and that it is our privilege as well as

our obligation to compensate them for the past injustices that have been forced upon them.

This is a most important branch of social work, for it not only gives aid to the clients themselves, but also promotes and develops ideas that will affect every young person from childhood on. It helps society toward a fuller and truer democracy than it can possibly attain with great and steadily increasing numbers of its older people repressed and depressed, as they are today.

Program of the American Geriatrics Society Fourth Annual Meeting June 5, 6, 7, 1947 Hotel Brighton Atlantic City, N. J.

THURSDAY, JUNE 5, 1947

8:30 P.M. Business meeting. Fellows invited.

FRIDAY, JUNE 6, 1947.
Walter E. Vest, M.D., Presiding

10:00 A.M. Bed Rest in Aged Patients. Albert Mueller-Deham, M.D., New York, N.Y. **Discusser:** Malford W. Thewlis, M.D., Wakefield, R. I.

10:30 A.M. Seventh Age of Man. Joseph B. Bolin, M.D., Norwood, Ohio. **Discusser:** Pierre J. Juhel, M.D., Cincinnati.

11:00 A.M. Preclinical Medicine and Geriatrics. Malford W. Thewlis, M.D., and E. T. Gale, M.D., Wakefield, R. I. **Discusser:** Albert Mueller-Deham, M.D., New York.

11:30 A.M. Functional Psychoses in the Aging Simulating Organic Syndromes. Edward B. Allen, M.D., White Plains, N. Y. **Discusser:** E. T. Gale, M.D., Wakefield, R. I.

12:00 Noon. Shakespeare, Gerontologist. Walter E. Vest, M.D., Huntington, W. Va.

FRIDAY, JUNE 6, AFTERNOON
Hollis E. Clow, M.D., Presiding

2:00 P.M. Tuberculosis in the Aged. Jay Arthur Myers, M.D., Minneapolis. **Discusser:** Arnold S. Anderson M.D., St. Petersburg, Fla.

2:30 P.M. The Use of Leisure Time in the Adjustment of Older People. Miss Helen Noyes, New York City. **Discusser:** Ruth Laverty, New York City.

3:00 P.M. Electric Shock Therapy in the Aging. Harold Lovell, M.D., New York City. **Discusser:** Edward B. Allen, M.D.

3:30 P.M. Public Health in Geriatrics. William F. King, M.D., Indianapolis, Ind. Discussion.

4:00 P.M. Personality Adjustment Following Hospital Treatment in Patient with Involutional Melancholia. Hollis E. Clow, M.D., White Plains, N. Y. **Discusser:** Harold W. Lovell, M.D., New York City.

4:30 P.M. Case Work on the Aged. Julius Weil, Cleveland Heights, Ohio. **Discusser:** Joseph I. Goodman, M.D., Cleveland, Ohio.

5:00 P.M. Practical Aspects of Geriatric Ophthalmology. F. L. Philip Koch, M.D., New York.

7:30 P.M. Dinner. Edward B. Allen, M.D., Presiding. Progress in the Solution of the Cancer Problem. William Seaman Bainbridge, M.D., New York. Presented by S. J. Turel, M.D., New York.

SATURDAY, JUNE 7, 1947
Edward B. Allen, M.D., Presiding

10:00 A.M. The Basal Ganglia as a Factor in the Process of Aging. Motion Pictures. S. Philip Goodhart, M.D., New York. General Discussion.

10:30 A.M. Institutions for the Aging: Their Present Program and Future Needs. Miss Ollie Randall, New York. **Discusser:** Edward B. Allen, M.D., White Plains, N. Y.

11:00 A.M. Coronary Thrombosis in Older Patients. Gabriel F. Greco, M.D., Ozone Park, N. Y. **Discusser:** Carl A. Williams, M.D., St. Petersburg, Fla.

11:30 A.M. Geriatric Public Health. Wingate M. Johnson, M.D., Winston-Salem, N. C. **Discusser:** William F. King, M.D., Indianapolis.

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GERIATRICS

Official Journal of the American Geriatrics Society

OFFICERS OF THE AMERICAN GERIATRICS SOCIETY

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Correspondence pertaining to the American Geriatrics Society should be addressed to Dr. Malford W. Thewlis, Secretary, 25 Mechanic Street, Wakefield, Rhode Island.

MEDICAL MEMORIAL FUND

There is one death a minute, day and night, in the United States, from diseases of the heart and arteries—yet comparatively little study of these diseases has been made in the past. Every day that we delay an adequately intensive study of cardiovascular diseases means about 1,500 additional deaths which may be unnecessary.

In 1940 only 17 cents per death was spent by the foundations for research on diseases of the heart and arteries. This is in contrast with the $6 per death spent on infectious diseases and particularly with the $500 per death spent on poliomyelitis.

The problems of aging are also of primary importance but have had, up until now, only negligible attention. In fact, medical research has been largely hit or miss, with attention focused on problems which have been the fad of the moment and which offered the possibility of quick dramatic results. Little consideration has been given to the relative importance of the diseases being studied.

To meet this situation a new foundation—Medical Memorial Fund, Inc. —has been created. Medical Memorial Fund receives its income through gifts from the public. These may be made as memorial gifts at the time of the death of friends or relatives. As the Fund receives each memorial gift, it sends a dignified memorial certificate to a relative of the deceased in accordance with instructions from the person making the gift.

This plan appeals to many people as an appropriate expression of sympathy—a lasting memorial of benefit to mankind. Here is an opportunity

for every individual to make a contribution within his means in support of important medical research. Every gift, whether $10 or $10,000, is helpful in the accomplishment of the purposes of the Fund.

The chairman of the board of trustees of the Medical Memorial Fund is Dr. Harlow Shapley of Harvard University, and the president is Dr. Henry S. Simms of Columbia University School of Medicine. Sound administration of the funds is guaranteed by a distinguished board of trustees and scientific committee.

Honorary members of the Lay Council include Chauncey Belknap, John W. Davis, Marshall Field, Herbert Hoover, and Albert N. Williams.

Members of the board of trustees are A. J. Carlson, Russell L. Cecil, John V. Duncan, Guy Emerson, Harry Goldblatt, Theodore G. Klumpp, Albert D. Lasker, Chauncey D. Leake, William Loeb, Currier McEwen, William deB. MacNider, Donald G. Price, Harlow Shapley, Henry S. Simms, J. Murray Steele, Lowell Thomas, Marvin R. Thompson, D. D. van Slyke, A. Ashley Weech, George H. Whipple, and Mark Woods.

The scientific committee is composed of Doctors J. Murray Steele, A. J. Carlson, Russell L. Cecil, Harry Goldblatt, Theodore G. Klumpp, Chauncey D. Leake, William deB. MacNider, Marvin R. Thompson, Henry S. Simms, D. D. van Slyke, A. Ashley Weech, and George H. Whipple.

Individuals who wish to make their contributions to the future health of the nation will welcome the establishment of this unique foundation for medical research. The offices are located at 650 West 165th Street, New York, 32, New York.

(Continued from page 187)

12:00 Noon. Sodium Pentothal in the Aged with Respect to Eye Surgery. William E. Froschauer, M.D., and Cyril E. Schrimpf, M.D., Cincinnati. **Discusser:** F. L. Philip Koch, M.D., New York City.

SATURDAY, JUNE 7, AFTERNOON
Wingate M. Johnson, M.D., Presiding

2:00 P.M. Management of Descensus Uteri in the Aged. Bernard L. Cinberg, M.D., New York. **Discusser:** Maurice Rashbaum, M.D., New York.

2:30 P.M. Endocrine Problems After the Age of Forty-five. Willard O. Thompson, M.D., Chicago. **Discussion.**

3:00 P.M. The Thrombosis—Embolism Problem Associated with Hip Fracture in the Aged. Harold F. Bishop, M.D., New York. **Discusser:** Donald L. Burdick, M.D., New York.

3:30 P.M. Treatment of Trauma in the Aged. Irwin E. Siris, M.D., New York. **Discusser:** John H. Mulholland, M.D., New York.

4:00 P.M. Geriatrics Contacts Old Age Assistance. Edward L. Tuohy, M.D., Duluth, Minn. **Discussers:** Jay Arthur Myers, M.D., Minneapolis, and Julius Weil, Ph.D., Cleveland Heights, Ohio.

4:30 P.M. Basal Metabolism in Persons Past Mid-Life. William B. Kountz, M.D., St. Louis, Mo. **Discusser:** Margaret Chieffi, M.D., St. Louis.

5:00 P.M. Orthopedic Problems in Old Age. Harold Lefkoe, M.D., Philadelphia. Discussion.

Digests FROM CURRENT LITERATURE

ORGANIZATION OF RESEARCH ON AGEING

(This is an excerpt from an English publication, *Old People, Report of a Survey Committee on the Problems of Ageing and the Care of Old People,* published for the trustees of the Nuffield Foundation by Geoffrey Cumberlege, Oxford University Press, London, 1947.)

The study of ageing is a special branch of the physiology, biochemistry, pathology, and psychology of the animal; but it is not in itself a self-contained subject. For this reason, perhaps, the study of ageing as a research problem remains ill-defined and unorganized. Little information is as yet available as to the characterization of the ageing process, or as to its causes and results. Still less is known of factors accelerating or retarding the process. Each of these problems can be approached by observational or experimental methods, statistical, biochemical, pathological, physiological, or psychological. Many specialist sciences are therefore involved. Of the many facets of the problem, some are likely to yield up important and useful information fairly quickly without much difficulty. Others could be examined only as part of a long-term programme of research, and might well involve years of apparently sterile work.

The characteristics outlined above indicate clearly the requirements of any organization designed to promote or initiate biological and medical research in gerontology. The organization should be such as to give coherence and standing to the subject, to formulate and define the main subdivisions of study, and to facilitate the fullest use of the techniques and data of relevant sciences. At the same time the organization must be such as to give the greatest possible flexibility of programme and personnel, so that promising lines can be followed quickly and vigorously by workers of suitable qualifications.

These considerations suggest that for the most part there should be no attempt at present to centralize research on ageing, and that the most effective way to promote research would be:

(a) The support over a period of years of research-units investigating specific problems of major importance.
(b) The establishment of a number of fellowships in gerontology, to be held for a period of years in suitable centres of research.
(c) The provision of short-term or part-time grants to suitable workers unable to undertake the full obligations of a fellowship.
(d) The provision of adequate research expenses grants to fellows, grantees, and others.
(e) The provision of bibliographic and other guidance to research

workers, if such a service comes within the general scope of the Foundation's activities.

(f) The use of the present Medical Sub-Committee or similar body to co-ordinate the whole research programme and to advise the Foundation on applications for fellowships and grants.

The following classification indicates the main problems for research on ageing:

Classification of Problems for Research in Gerontology

A. Characterization of the process of ageing.
   1. Morphological changes.
      (a) Gross changes in organs and systems.
      (b) Histological changes in organs.
   2. Functional changes.
      (a) Changes in normal psychological function.
      (b) Changes in normal physical function.
      (c) Pharmacological changes with ageing.
      (d) Changes in relation to injury.
      (e) Immunological changes.
   3. Biochemical changes.
      (a) Changes in body composition.
      (b) Changes in biochemical function.
   4. Incidence and severity of physical and psychological disease at different ages.

B. Factors which may accelerate or retard or alleviate ageing.
   1. Heredity.
   2. Nutrition.
   3. Physical and psychological environment and fatigue and rest.
   4. Hormonal and other special deficiencies or excesses.
   5. Specific diseases.
   6. Early detection and treatment of disease.

The schemes of research so far considered by the Medical Sub-Committee cover but a small part of the wide range of problems requiring investigation.

ERRATUM

In the article, "Physical Fitness and Work Capacity of Older Men" by Ernst Simonson (March-April), the second and third lines from the bottom on page 116 should be omitted. The last line on page 118 should read, "Also age trends of sickness and accident absenteeism are important. A rather large material on absenteeism has been accumulated but cannot be reviewed within the scope of this article. The discussion was restricted" . . . etc., to the end.

Surgical Treatment of Acute Cholecystitis in the Aged.

SEBASTIAN J. CARNAZZO. *J. International College of Surgeons,* 9, No. 6:764-73 (No. V-Dec.), 1946.

Acute cholecystitis occurs rather frequently in individuals over 55 years of age, so that it is essential that this problem should be given special attention. Cholecystitis must be considered as the exacerbation of a chronic process of long duration. It has been ascertained that the incidence of general organic disorders, such as diabetes mellitus, arteriosclerosis, chronic nephritis, cardiac trouble, hypertension, renal and pancreatic lithiasis, and arthritis, is greater in individuals suffering from disorders of the bile duct.

Attacks of acute cholecystitis in patients over 55 years of age require urgent and careful surgical attention. The peculiarities and extent of the clinical problem, the application of adequate surgical therapy, as well as the treatment of existing general organic disorders, should be attended to with utmost care. With pre- and postoperative therapy, safe anesthesia, and operative technique, such patients may be operated on with lowered percentage of mortality. Cholecystectomy is the operation of choice.

During the intervention all patients received intravenous injection of either a solution of dextrose (10 per cent in distilled water), or blood or plasma transfusion. Penrose drains were introduced into Morison's pouch and two drains into the area of the cystic duct. Powdered sulfanilamide and sulfathiazole, 2 gr. of each, were entered into the peritoneal cavity. Postoperatively, at first, 100,000 units of penicillin were administered intravenously every twelve hours during several days, followed by intramuscular injection of 25,000 units every three hours during five or six days. Levin's tube was generally removed on the third day after the operation.

Ten figures.

Care of the Elderly.

LORD AMULREE. *The Lancet,* 2, No. 22:801–2 (Nov. 30), 1946.

How to give the elderly their proper place as essential members of the community will probably be the great social question of the future. The change in the increase of average expectation of life makes it likely that in the next fifty years patients admitted to hospitals will be drawn in ever-increasing numbers from the higher age groups.

The question of providing adequate living quarters for the aged is most important. In 1944 there were over 500,000 British pensioners living alone; 85,000 were over 80 years old. This loneliness is one of the most appalling aspects of old age.

In projected buildings for old people the rooms should be on the ground or first floor. The staircase must be well lighted and have a handrail on each side. Ten per cent of fatal domestic accidents to persons over 60 are traced to accidents on the stairs. Floors should be of nonslip material. Handchains over the baths are recommended. Fireplaces should be protected; 5 per cent of fatal accidents to the aged are due to burns.

A living room with a curtained bed recess, bathroom, and kitchen are sufficient. In hostels as much privacy as possible should be provided; the beds can be arranged with screens, so that the inmates can retire. Cubicles with a door, as in the Royal Hospital, Chelsea, containing the bed, chest of drawers, and chairs, have been found very convenient.

France has exemplary institutions of this type; larger bed-sitting-rooms are available at £1 per week, but there are also smaller rooms and wards. Food is served in a common dining-room.

Hypertension; Some Uses and Abuses of the Thiocyanates.

M. HERBERT BARKER. *Texas State J. of Med.* 42. 462-465 (Dec.), 1946.

Praise and denunciation of thiocyanates appear in clinical literature. The carefully controlled individualized dosage necessary to maintain the desired thiocyanate concentration in the body, between 6 and 12 mg., can be directed by the determination of the blood-cyanate concentration, as published in 1936 by the author, thus avoiding overdosage.

Use of thiocyanate is indicated in: (1) Plethoric hypertension (benign or essential) with distressing symptoms and/or cardiovascular disturbances in which the red cell pack is usually found to be over 50 per cent. Continued controlled blood cyanate concentration (8 to 12 mg. per 100 cc.) usually results in prompt relief of cerebral symptoms and gradual decrease of systolic blood pressure of 10 to 20 mm. per week. Rarely should the total cyanate dosage exceed 5 grains per day in the beginning months.

(2) Uncontrolled congestive heart failure in hypertension, where digitalis is insufficient. If the systolic and diastolic pressure may be reduced to 150/90, a patient may recover from severe repeated decompensation to return to a surprisingly satisfactory sustained activity. One patient, now aged 81, continued cyanate therapy for 13 years with a blood concentration between 8 and 12 mg.

(3) Patients having sympathectomies for their hypertension should be offered controlled thiocyanate therapy if distressing manifestations are present.

(4) Chronic glomerular nephritic patients with hypertension may obtain symptomatic relief and regain circulatory compensation by similar blood concentrations of thiocyanates. Reductions of blood pressure from 220/140 to 120/90 have been observed.

Thiocyanates should be stopped during febrile periods of infections and prior to surgical procedures. Sedatives are better omitted during the initial week of thiocyanate therapy, so that symptoms may not be confused or clouded.

One figure. Bibliography of 15 references.

Hypertension and Calcium Intake.

J. HARRIS. *The Lancet* (London) 2, No. 23:849–50 (Dec. 7), 1946.

Calcium administration per os has a blood pressure raising action; however, it does not follow from this, that it is a causal factor in hypertonicity.

According to E. V. Cowdry, London (1933), the affluent classes whose intake of calcium is unusually high, show a prevalence of arteriosclerosis and allied conditions. A patient may have arteriosclerosis without hypertonicity, and vice versa, but hypertonicity cannot be diagnosed by x-ray examination. The latter does not lend itself to demonstrate a moderate increase of arteriosclerosis as the method is not exact enough for such a purpose.

In rabbits it has been demonstrated by the author, through analyzing the tissues of the aorta, that a high calcium intake enormously increases the calcium content of these vessels. Heubner found, on increased calcium intake by test animals, that arteries and tendons contained more calcium than other tissues.

By adding calcium to bread, the authorities are conducting a large experiment on forty million human beings. Everybody admits that millions of people do not need the added chalk. The possibility of their suffering injury by it cannot be excluded. As a matter of fact, it is certain that an intake of such a potent substance as calcium in unnecessary quantities must inflict injury. If calcium is retained, it is bound to do harm; if an unnecessary amount of calcium passes out via the kidneys, it causes unnecessary renal strain. There is not a shadow of

doubt that the additional chalk must in many instances cause injury and shorten life.

Medical Management of Hypertension.

WOODFORD B. TROUTMAN. *Kentucky M. J.* 45. No. 1:7-8 (Jan.) 1947.

In many instances hyperpiesia may be a physiological response or requirement to maintain proper body function. Author is of the opinion that the elderly person with a blood-pressure of 180/80 and marked arteriosclerosis needs this blood pressure. When the diastolic level of the blood pressure is elevated, then we have a true hypertension, and improvement should be primarily gauged by the amount of reduction we can get in this phase of the pressure.

One should think more in terms of etiological factors in planning the attack on a given case of hypertension. Griffith speaks of five types: (1) The renal type, subdivided into renal ischemia, for which there is no effective treatment; beginning renal failure, for which vitamin A may help, before the blood urea goes up; renal infection with treatment for the infection. (2) Pituitary over-activity, in which case irradiation of the gland may help. Also pitressin tannate in oil once a week for two months, later once monthly. Testosterone and progestrone may be helpful. (3) Adrenal type, requiring surgery for tumor. (4) The type in which there is increased fragility or permeability of the small blood vessels, for which Rutin is beneficial. It has no effect on the blood pressure but there is less danger of hemorrhage and apoplexy. (5) Miscellaneous types, including lead poisoning, etc.

General therapy: rest with proper recreation and moderate exercise; if the patient is obese, he should lose weight; use of tobacco should be stopped if at all possible; administration of sedatives which are palliative but do not exert lasting benefit, nitrites have little if any value; thiocyanates are believed to be the one drug of value, aside from sedation. Author uses 2-3 grains daily. Garlic and the like seem to be worthless. Salt-free diet may help to relieve the signs of congestion.

Medical measures should be given a fair trial, and if they are of no avail, the properly selected cases should be offered the benefit of surgery. The patient should be informed that it may possibly add several years to his life, and then let the individual make his own choice.

Hypertension: Results of Splanchnicectomy in Patients with Abnormal Pyelograms.

REED M. NESBIT and ROBERT T. PLUMB. *J. Urol.* 57. No. 1:116-119 (Jan.) 1947.

Splanchnicectomy resulted in symptomatic improvement in most of the sixteen patients (13 females and 3 males) herein reported, and corresponding lowering of the elevated blood pressures occurred. All of the patients observed had structural abnormalities of the kidneys which were made evident by pyelography; some were bilateral and others unilateral, and splanchnicectomy appears to have caused no improvement in one female patient only whose renal disease was already too far advanced, because she died two months later of uremia.

It appears that gross renal disease does not in itself prevent an effective response to splanchnicectomy, but the operation in question should not be advocated when the prognosis for life is adversely affected by advanced kidney disease.

2 tables and 1 figure with pyelograms in six patients.

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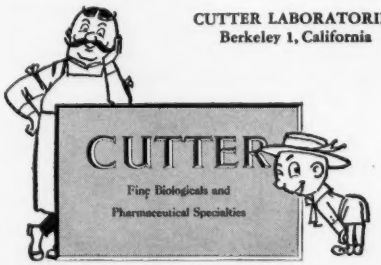

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Thoracoplasty for Pulmonary Tuberculosis; Observations on 32 Patients in the Older Age Group.

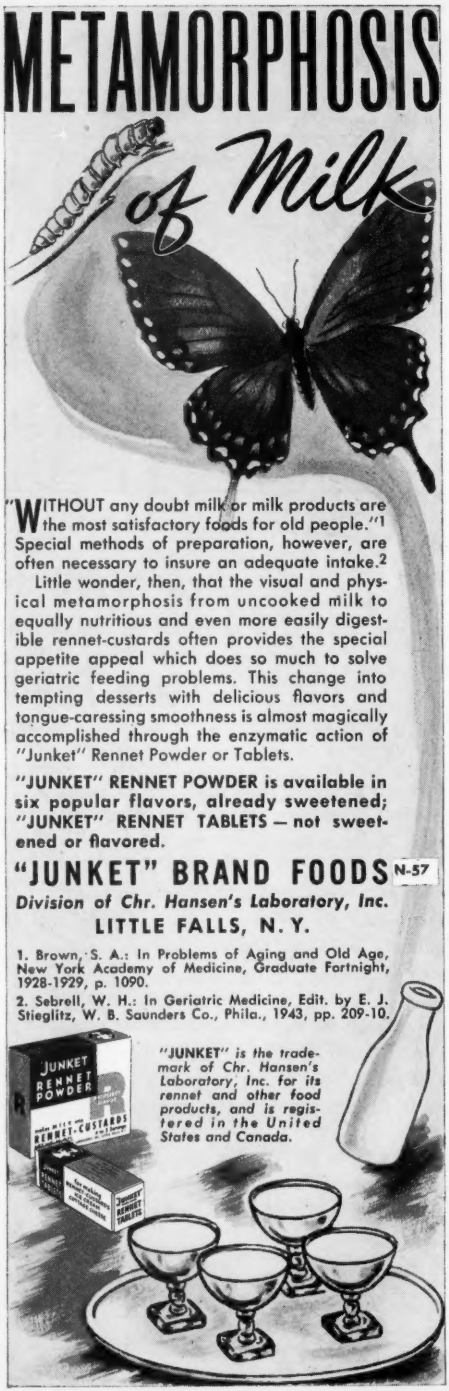
PAUL E. ZUELKE; W. E. ADAMS, and ROBERT G. BLOCH. *American Revue of Tuberculosis* 54, No. 4-5; 368-375 (Oct.-Nov.), 1946.

The results following thoracoplasty for pulmonary tuberculosis in 32 patients between 40 and 65 years of age are presented. The operations were all done under ethylene plus oxygen anesthesia. Preoperative medication consisted of 0.18 g. calcium nembutal and 0.01 morphine sulphate. The patients were all awake before leaving the operating room. The cough reflex was thus depressed for as short a time as possible. Long segments of three ribs were usually removed at each posterior stage. One-half of the patients received anterolateral supplementary operations in addition to the paravertebral stages (mostly two paravertebral stages; in four cases, however, three). The next stage usually followed in two weeks, in order to obtain the maximum amount of collapse before there was regeneration of bone in the beds of the ribs previously resected. In only two cases it became necessary to remove more than seven ribs. Blood transfusions were used freely.

The operations under consideration compare favorably with those reported by Dolley, Jones and Paxton from fourteen different institutions in patients of all age groups. This substantiates the authors' view that the age of the patient *per se* should not be the determining factor in the choice of therapy. Patients over 40 were able to tolerate the operation and were benefited as much as those under that age.

Thus it would seem that thoracoplasty should be used more frequently because of the increasing age of our tuberculous population, and because so many of these older patients cannot be cured by the more conservative methods of collapse therapy.

Eight tables; bibliography of 6 references.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1. Brown, S. A.: In Problems of Aging and Old Age, New York Academy of Medicine, Graduate Fortnight, 1928-1929, p. 1090.
2. Sebrell, W. H.: In Geriatric Medicine, Edit. by E. J. Stieglitz, W. B. Saunders Co., Phila., 1943, pp. 209-10.
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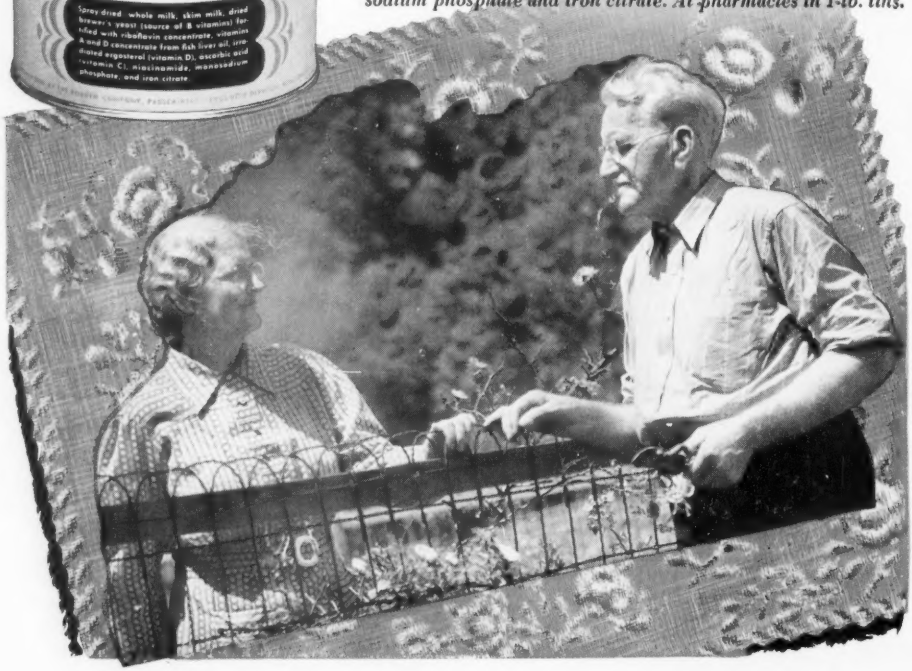
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